DOCTOR OF PHILOSOPHY

A partnership between health visitors and dentists to identify high caries risk Scottish pre-school children

Ballantyne-MacRitchie, Heather

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DUNDEE UNIVERSITY

To Mum, John and Iona

and in memory of my Dad

"..we also rejoice in our sufferings, because we know that suffering produces perseverance; perseverance; character; and character, hope."

" people who say it cannot be done should not interrupt those who are doing it"

Dan Clark

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Declaration

This thesis is a record of research I have undertaken at the University of Dundee, and have written by myself. It has not previously been submitted for a higher degree. I have consulted all references cited.

Heather M Ballantyne-MacRitchie

Certificate

I hereby certify that this candidate has fulfilled the relevant Ordinance and Regulations of the University of Dundee for the degree of Doctor of Philosophy.

Professor N B Pitts

Supervisor

Dr. C Longbottom

Supervisor

Abstract

The study forming the basis of this thesis was designed to examine the feasibility of a partnership between health visitors and dentists to access pre-school children for data collection which could allow identification of those children at risk of developing dental caries. The purpose of the investigation was twofold: 1) to investigate the feasibility of a partnership between dentists and health visitors (existing health services personnel) to access pre-school children in order to collect dental, microbiological, health behaviour and socio-demographic data at ages 1, 2, 3 and 4-years and 2) to develop a novel caries risk assessment model (using such data) for the identification of 4-year old pre-school children at high risk of developing dental caries.

The basis for the investigation was a prospective 4-year longitudinal study of consented children from age 1- to 4-years inclusive. This was the first, large scale longitudinal study of pre-school children to involve a consented, but non-exclusive, population cohort. The cohort comprised all those children born and resident in Dundee between 1 April 1993 and 31 March 1994 for whom written consent was obtained by the child's health visitor at 8-months of age (n = 1683). Health visitors obtained microbiological (saliva sampling) and socio-demographic (parental and health visitor questionnaires) data in partnership with a study dentist collecting dental data (dental examination).

The results of the study suggest that health visitors could, within their daily

caseload of duties, both access the majority of pre-school children and, independently, collect caries risk assessment data relating to these children with sustained diligence over a 4-year period

Risk model development was carried out using both logistic regression and CHAID (Chi-squared Automatic Interaction Detector) analyses. Data collected at age 1-year was used to predict caries at age 4-years. This resulted in the development of the Dundee Caries Risk Model (DCRM) (sensitivity 69% and specificity 60%) (n = 784). The key predictive factors in this model were type of housing, use of a feeder cup and use of vitamins. It might have been assumed that microbiological factors would be of significance. However, they were not found to be sufficiently predictive for incorporation into the model. This reduces the cost and increases the simplicity of the risk model.

Development of the DCRM may facilitate preventive care being targeted towards those at risk of developing dental caries in order to prevent overt manifestation of caries in pre-school populations.

Chapter 1: General Introduction

1.1 Dental Caries

1.1.1 Overview of the disease process

Dental caries (from the Latin *caries*, decay) simply means decay or rotting of the teeth and is the process of tooth decay (Johnson, 1991). What is now referred to as caries is not a single event, but rather the outcome of an accumulation of events, a process, extending over a period of time (Fejerskov, 1997). This process is a highly dynamic one, with alternating periods of destruction and arrest (or even repair). When the destructive forces predominate the disease will progress and lead to irreversible breakdown of hard tissues. Conversely, preventive measures, such as dietary control, effective plaque removal and judicious use of fluoride, can arrest the disease process and, provided this occurs prior to cavitation of the lesion surface, partial repair is possible. Consistent with the dynamic nature of the carious process, cycles of active disease and arrest of caries lesion progression are common (Manji et al 1991). Evidence tends to support the notion, however, that true remineralisation is a very rare phenomenon *in vivo* (Larsen & Fejerskov, 1989) and clinical experience suggests that arrested lesions may remain as lifelong whitish or brownish caries "scars" in the tissue (Nyvad and Fejerskov, 1997). An oversimplified but essentially accurate concept of the aetiology and pathogenesis of dental caries has existed for a century and has come to be known as the acidogenic theory, propounded initially by W.D Miller (Miller, 1890). This theory holds that bacteria present in the mouth interact with retained food particles to produce substances

capable of dissolving enamel. The three essential components of the carious process are thus immediately appreciated, namely the presence of a susceptible tooth, the presence of certain types of microorganisms and dietary factors. Many other factors, both local and systemic, influence the likelihood of caries developing and its speed of progression, hence caries can be seen to be a multi-factorial disease. The initiation of a carious lesion at a given tooth surface, be it enamel of a crown or the cementum of an exposed root, is customarily explained as a series of physico-chemical phenomena in which acids produced by the metabolism of dental plaque produce subsurface demineralisation of hard tissue. Progress, arrest, or repair depends on the balance of relevant physico-chemical factors, such as the solubility of tooth mineral, the local pH and ionic concentration of the environment at the tooth surface. Dental caries is thus a complex, dynamic biological process. It cannot be defined in terms of a single event or observation (such as a cavity on a particular tooth surface), or in terms of a substance (such as dentine demineralised and/or infected as part of the process).

The earliest macroscopic evidence of caries is known as the 'white spot lesion'. Such lesions form in areas of plaque stagnation such as (1) enamel pits and fissures in the occlusal surface of molars and premolars, (2) immediately cervical to the contact areas of approximal enamel smooth surfaces and (3) at the enamel of the cervical margin of free smooth surfaces adjacent to the gingival margin. Lesions may also appear brownish in colour, those with intact surfaces being described as 'brown spot lesions'. The extent of discolouration or staining is dependent upon the degree of exogenous material adsorbed by the porous enamel. (It has been noted

some years ago in Switzerland that lesions with dark brown or black discolouration of small smooth surface lesions showed in almost every instance a cavity (Marthaler and Germann, 1970)). Eventually, if the lesion progresses, the porous, but relatively intact surface breaks down (cavitation) and a hole is formed (cavity). Not all lesions, however, progress to cavitation. There is evidence to suggest that these 'pre-cavitated' lesions are now more prevalent than cavitated lesions (Ismail, 1997) and this has an important impact on the diagnosis of dental caries and delivery of patient care. The carious lesion in enamel has been divided into zones based upon its histological appearance (Silverstone and Hicks, 1985). Four main zones may be distinguished. There is a translucent zone, a dark zone, body of the lesion and finally the surface zone, which is relatively unaffected. Beyond the enamel, the pulp and dentine of the tooth are integral parts of the same living tissue complex. This dentine/pulp complex is a vital tissue capable of defending itself and the progress of caries in dentine involves a fluctuating interplay between attacking forces and defence reactions. The state of the tissue at any specific time depends, therefore, on the extent of each of the two processes. An explanation of the defence reactions of the pulp-dentine system and the numerous changes in dentine both before and subsequent to enamel cavitation are outwith the scope of this thesis and the reader is referred to relevant texts (Silverstone et al, 1981 and Thylstrup and Fejerskov, 1994).

1.1.1.1 Long term impact of dental caries

In addition to personal suffering, the almost universal presence of caries in western communities presents society as a whole with a considerable problem. However financed, the cost to the community is large. Only if a much greater emphasis is placed on prevention will individual suffering be reduced and the cost to society be kept within practicable limits. For the purposes of this thesis, the focus has been placed on the impact of caries on children, more specifically pre-school children. Many children suffer pain and trauma, both physical and emotional, due to the consequences of caries. In Scotland, 41.4% of those 5-year olds with decay have unrestorable or missing teeth with the resultant additional sequelae of space loss and crowding. (Pitts et al, SHBDEP Report, 1997/98). Preventive measures implemented at the earliest possible stage in child development could assist limitation of this suffering and could also contribute very significant overall benefits when its impact on lifelong caries experience is considered e.g. the fill / refill restorative cycle.

1.1.2 Thresholds of caries diagnosis

The carious process is not inevitable. An important role for the dentist, therefore, is early diagnosis of disease and where possible, prevention of progression and recurrence. At present, there is no caries diagnostic tool in current clinical use which fulfils all of the ideal criteria and at a population level these tools significantly underestimate overall caries experience (Pitts, 1997). Ideally a diagnostic tool would be: non-invasive; provide simple, reliable, valid, sensitive, specific and robust

measurements of lesion size and activity; based on biological processes directly related to the caries process, while being affordable, acceptable to dentists and patients; and capable of early implementation into both clinical practice and research settings. A review of caries diagnostic methods is outwith the scope of this thesis and the reader is referred to relevant texts (Pitts, 1991(a), Pitts, 1991(b), Pitts, 1992, Longbottom, 1992, Lussi, 1993, Angmar-Mansson and Ten Bosch, 1993, Stookey, 1996, Verdonschot et al, 1999)

In order to describe the thresholds of caries diagnosis used in this study it is helpful to consider the totality of the carious process as an iceberg (Pitts, 1994) (Appendix 1.1). The diagnostic threshold employed will determine the level at which the iceberg floats. The threshold used in classical epidemiological studies is that of clinically detectable lesions into dentine – this threshold is called "D_3" in World Health Organisation terminology (D, denotes permanent teeth and d, deciduous teeth). The use of this D_3 threshold means that all the signs associated with caries that are less severe than clinically detectable dentinal lesions are ignored and considered as "caries-free". The iceberg has been divided into several discrete levels or diagnostic thresholds graded from the most severe D_4 (lesions extending into the pulp chamber) to sub-clinical lesions smaller than even the clinically detectable D_1 lesions (enamel caries with apparently intact surfaces). The present study used the diagnostic threshold of d_1, caries into enamel threshold, i.e., all detectable enamel caries comprising lesions at $d_1 + d_2 + d_3 + d_4$. It should be noted that examination for caries undertaken in different and more optimal clinical settings

might have detected more lesions than in the present 'field' setting (the methodology of caries diagnosis for this study will be discussed fully in Chapter 6.5.1.1.).

1.1.3 Scale of the caries problem and distribution within the population

The prevalence of caries increased steadily with the advance of civilisation and dental caries and periodontal disease are the most common diseases affecting western man (Silverstone et al, 1981). Studies have shown, however, that there are differences in trends between developing and developed countries, and some have suggested that we could be in the early stages of a global epidemic of dental caries, since 80% of the world's population lives in developing countries (Johnson, 1991). Johnson also noted that data from many countries show that disease levels in any given age group are by no means normally or evenly distributed. This means that, although the overall prevalence of caries has gradually reduced in the developed world, the majority of the disease is often found in a minority of the population, with large proportions apparently "caries-free". (Pitts, 1997). Focusing on children, the 1993 U.K children's dental health survey, carried out by the social survey division of the Office of Population Censuses and Surveys (OPCS), stated that since the previous survey more than ten years before, the proportion who had decayed or filled primary teeth at the time of the dental examination decreased for children of all ages from five to eleven years (O'Brien, 1994). However, despite this improvement, in 1993, over three fifths of nine year old children in the United Kingdom had one or more primary teeth with known decay experience. The

improvement in the proportions of children with no known decay experience in the primary dentition was greater among eight to ten-year olds than among the younger children. Among five-year olds, for example, the proportion of children with actively decayed and/or filled teeth decreased from 50% to 45%, while among nine year olds it fell from 71% to 61% (OPCS 1993). It was also shown that 46% of the 5- year olds had all the decay. For this thesis the focus was placed on the dental health of Scottish children. This is currently assessed by the Scottish Health Boards' Dental Epidemiological Programme (SHBDEP, 1997/8, Pitts et al, 1998) and is a joint venture between all fifteen Health Boards and the Chief Scientist Office's Dental Health Services Research Unit based at the University of Dundee. Caries is recorded at the d_3 level. The prevalence of dentinal decay in Scotland is much higher than in England and Wales and the most recent survey for 5-year olds (SHBDEP 1997/98) showed a mean $d_3$mft of 2.7 per child, considerably higher than the mean of 1.8 for Great Britain, which was recorded in the BASCD surveys of 1995/96 (Pitts & Evans, 1997) and previously in 1993/94 (Pitts and Evans, 1995). These surveys also emphasise the uneven distribution (or skew) of decay within the population, with only 43% of the five year olds apparently "free" of dental decay experience at the caries into dentine level of detection (d_3). All the decaying teeth were found in just 52% of the children. Some of these children had very high disease levels and more than half of the untreated decay was found in just 9% of the children - these children can, therefore, (retrospectively) be deemed to have been 'at risk' or at 'high risk' of developing dental caries. For the purposes of this thesis, 'at risk' relates to any 4-year old pre-school child with caries ($d_1$mft > 0, $d_3$mft > 0), in

comparison to 'high risk' which denotes a $d_1mft \geq 3$ or $d_3mft \geq 3$ at 4-years of age (see chapter 4.4.5). When compared to the five previous surveys, the latest data appear to add to the overall picture of a bottoming out of the decline in caries prevalence seen in the eighties (SHBDEP, 87/88, 89/90, 91/92, 93/94, 95/96). These figures form the basis for the rationale behind the investigations reported in this thesis. The implications of early identification of these high risk children, in terms of the potential benefits for the children themselves and, in the wider sense for the community and health services as a whole, are enormous.

1.2 Need for identification of at-risk individuals

The ability to detect in advance of overt manifestation, those at high and low risk of developing dental caries has many potential advantages for the patient, the clinician and public health planners. It would allow preventive efforts to be focused on those individuals or groups of individuals most at risk of developing caries, in a cost-effective fashion, without, of course, reducing the community wide benefits of mass preventive methods (such as water fluoridation) – a so-called "twin-track" approach. In order to deliver primary prevention in this manner it is necessary to be able to accurately identify those susceptible individuals early, for example at 12-months of age, rather than waiting to measure irreversible damage at 5-years of age. If the caries incidence of the 10% minority at high caries risk in zero to 5-year olds could be reduced by half, the restorative treatment need for this zero to five-year old population would be reduced by around 25%. This underlies the importance of finding accurate predictive methods that can, with high sensitivity and specificity,

identify an individual at high risk of developing caries (see section 2.2.2 for statistical considerations). As noted, the levels of decay prevalence in Scottish 5-year olds remain unacceptably high (SHBDEP, 1997/98) and this was identified as a priority area by the Scottish Office in the policy statement 'Scotland's Health: A Challenge to us all' in 1992. At this time, a target was set for a reduction in dental caries in 5-year olds and this was re-stated in the 'Oral Health Strategy for Scotland' document, issued in December of 1995. This target stated that '60% of 5-year old school entrants should have no cavities, fillings or extractions by the year 2000'. A recent report (Scottish Office, 1998) admitted that it seemed unlikely that this target would be achieved and indeed a recent government white paper extended this target to the year 2010 (The Scottish Office Department of Health, 1999). It is obvious that the figure of 60% caries-free is well above the latest SHBDEP data of 43% of 5 year olds caries-free in 1997/98. Achievement of this current challenging goal for 5-year olds will require an emphasis on preventive based action. This preventive care should be focused on those individuals at highest risk and be implemented at the earliest possible age.

1.2.1 Prevention in the pre-school child

The ultimate goal of primary prevention is to change behaviour or alter a factor or factors in the environment so that dental disease is prevented from developing. The term 'control' is used by some workers in preference to prevention, to emphasise the importance of a continued programme of monitoring rather than a single preventive measure. The belief is held that dental caries cannot be totally prevented, but only

controlled (and monitored), so that lesion progression into the stage of frank cavitation is prevented (Fejerskov, 1997). There is much controversy at present at to what represents the first stage of the carious process and only when international agreement is reached on this important topic will clarification of the term prevention or control be recognised. This thesis will, therefore, use the term 'prevention' to represent a state where enamel (and hence consequent) carious lesions can be prevented from occurring (primary prevention), arrested (secondary prevention) or repaired and subsequent lesions prevented (tertiary prevention).

The levels of dentinal decay already present in 5-year olds in Scotland indicate that preventive programmes beginning in primary schools are targeted too late, as the disease process is already well established. To improve this situation a concentration of efforts, for a sustained period, on pre-school children and their parents/principal carers was recommended by the Scottish Office. This was in order to bring about not only a prevention of disease but also a positive attitude toward oral health (Oral Health Strategy for Scotland, 1995). The main areas recognised for action include: diet; health promotion; fluoridation and role of the individual. A brief description will be given for each of these but a comprehensive discussion of these procedures is outwith the scope of this text and the reader is referred to relevant texts (Murray, 1990 and Pine, 1998).

1.2.1.1 Diet

Many sources have demonstrated that there is a clear association between dietary sugar and dental caries (Holloway, 1988, Edgar and Higham, 1991, Rugg-Gunn,

1993). This is a complex issue, however, and although the weight of evidence supports this view, some authors have not reported such clear associations (see chapter 2.2.5). On ingestion of sugars, acid is rapidly generated in the dental plaque and within 1-2 minutes plaque pH has fallen to levels at which enamel dissolution can occur (Stephan, 1944; Fejerskov, 1992 and Macpherson, 1994). Dietary control in the prevention of dental disease in the pre-school child should be aimed at the parent or guardian in control of the child's daily dietary habits (The Scottish Office, 1995). Pre-school children are identified in the Government's White Paper *The Health of the Nation* (HMSO, 1992) as a key group. It notes that 'the adoption of healthy lifestyles during childhood encourages optimum growth and resistance to ill health, both emotional and physical'. There is increasing evidence to suggest that there is a relationship between growth and development, starting from before birth and during childhood, and risk later in life of coronary heart disease, raised blood pressure and poor oral health (Gregory et al, 1995). Advice should, therefore, be given on the importance of a good balanced diet and both the frequency and timing of refined carbohydrate intake. The problems of diet counselling are formidable, however, and often require the family to undergo a fundamental change in both attitude and behaviour. To have any chance of success, the methods used in diet counselling should be planned not only to give information, but also to persuade the parent, guardian and child to act on this information (The Scottish Office, 1995). A very informative booklet issued by the Health Education Board for Scotland (HEBS) outlines advice given to parents/guardians (HEBS, new Birth to Five, 1994).

1.2.1.2 Health Promotion

Health promotion has a key role to play and can be delivered to children and their carers in nursery schools, in the home, child and family centres, health centres and community centres. More importantly, all key health services personnel in contact with pre-school children should be encouraged to teach parents/guardians the importance of toothbrushing, early dental registration and use of fluoride toothpaste, as well as delivering essential dietary advice. The role health visitors play in this regard will be discussed in detail in Chapter 3. Other key personnel who could deliver health promotion are general medical practitioners and pharmacists. Of the utmost importance, however, is the consistency of the message (Stephen and Hesketh 1996)

1.2.1.3 Fluoridation

Prevention of decay in the deciduous dentition can be achieved by the use of topical fluorides, including varnishes, gels, solutions and, most commonly toothpaste (Murray, 1990). In the longer term, systemic fluoride in the form of tablets or drops (or more rarely fluoridated milk or salt) can be administered to the pre-school child to allow the benefits to be obtained for the permanent dentition. Fluoridation of the public water supplies is the single most effective measure which can be taken to prevent decay in the permanent dentition (Murray, 1990, Jones and Lennon, 1998). It is, however, an emotive issue and liable to be the centre of political arguments well away from objective evidence.

1.2.1.4 Role of the individual

In terms of pre-school children, the parent/guardian has the responsibility for the oral health of the children in their care. They can ensure that children learn how to brush their teeth and that they limit the consumption of sugary foods, snacks and drinks in their diet. They can also ensure that their child is registered with a dentist, at least as soon as the first teeth appear, and that the child has regular dental visits thereafter (Oral Health Strategy for Scotland, 1995), i.e. following guidelines of health promotion. The basis on which individual parents are equipped to take on this responsibility varies, as do their preferences and abilities.

1.2.2 Targeting of pre-school children

The prevalence and uneven distribution of decay in Scottish 5-year olds was outlined earlier in this chapter. Focusing preventive care on the 'at risk' minority, prior to age 5, would result in a more effective use of health care resources and could also minimise unnecessary emotional trauma to children both in the short and longer term. In addition, it would help reduce the cost of initial and repeated restorative care for these children, as well as reducing the provision of unnecessary preventive measures for low caries risk children, potentially resulting in financial savings for the National Health Service (NHS). Targeting an at-risk group, however, requires its prior identification. It is the aim of this thesis to explore the feasibility of working in partnership with health visitors in order to access pre-school children for the purpose of collection of caries risk assessment data to enable the identification of those at high risk of developing dental caries.

1.3 General research questions

The two main research questions to be answered in this thesis are:

1. Can a study dentist work in partnership with health visitors to gain access to a consented cohort of pre-school children for the purpose of caries risk assessment?

2. Can pre-school children at high caries risk be identified (through such a partnership) using dental, microbiological, dietary, oral hygiene, social, medical factors and hunch factors?

1.4 General hypotheses to be tested

To answer these research questions two general hypotheses proposed for the study will be tested. Further, more specific, hypotheses to be tested will be dealt with in the relevant individual chapters.

Hypothesis 1.1

It is **feasible** to employ existing health services personnel to access pre-school children in order to collect caries risk assessment data for a 4-year longitudinal caries risk assessment study in partnership with a study dentist.

Hypothesis 1.2

It is **feasible** to develop a multi-factorial caries risk prediction model in order to identify (to a reasonable degree of sensitivity and specificity) pre-school children at high risk of developing dental caries.

1.5 Outline of thesis

This thesis concerns the feasibility of a partnership between a study dentist and the health visitors of the city of Dundee to access and carry out caries risk assessment on pre-school children. Following a general introduction in this chapter, chapter 2 introduces the discipline of caries risk assessment, including a review of work carried out in this field, as well as providing an outline of the aims of the present study. Health visitors, their role and contribution in the health care community, will be discussed in Chapter 3, as well as the results required to determine the feasibility of working in partnership with these health care personnel. Chapter 4 details the methodological techniques used to collect the relevant data for risk assessment and the statistical tests used in this thesis. The results of the study are provided in Chapter 5. Chapter 6 consists of a general discussion of the importance and interpretation of the findings and subsequent recommendations. Finally, to conclude, Chapter 7 deals with the hypotheses tested and general conclusions drawn from the study as a whole.

Chapter 2: Caries risk assessment

2.1 Introduction

In this chapter caries risk assessment in pre-school children, including a review of the work carried out in this field, will be described. The chapter will conclude with the outline and aims of this thesis.

2.1.1 Definition of caries risk assessment

Risk is often defined as the probability of an event occurring within a specified period of time (Hausen, 1994). For the purposes of this thesis, caries risk was, therefore, the probability that a *pre-school child* would develop one or more carious lesions over a specified period of time. High caries risk was defined as a $d_1$mft of or $d_3$mft of greater than or equal to three at 4-years of age (clarification of this cut-off point for high risk has been provided in chapter 4.4.5). Caries risk assessment was defined as the assessment of the caries risk status of a *pre-school child*, in this thesis, a 4-year old child. Caries in pre-school children will be described as early childhood caries rather than use of the terms 'baby bottle decay or nursing caries'. The report of a recent international workshop (Drury et al 1999) defined the term "early childhood caries" as the presence of one or more decayed (non-cavitated or cavitated lesions), missing (due to caries), or filled tooth surfaces in any primary tooth. The participants recommended that the term "severe early childhood caries" refer to children with "atypical," "progressive," "acute" or "rampant" patterns of dental caries. The author of this thesis is in agreement with this definition. It is accepted,

however, that other terminology has been used to describe specific patterns of decay in pre-school children (Veerkamp and Weerheijm, 1995).

Many individual factors may be used either singly, or in combination in the assessment of caries risk. These may be known as **risk markers**, i.e. factors that are associated with the outcome, but for which the relationship does not need to be a causal one, or **risk factors** for which a causal association with the outcome has been established (Johnson, 1991). For the purpose of this thesis, both risk markers and risk factors were collectively termed risk *markers.* In any review of caries risk assessment the distinction between association (identification of risk markers) and prediction must be established. Hausen et al (1994) summarised the main distinctions and outlined the differing statistical approaches to each (statistical considerations will be dealt with in 2.2.2). They concluded that even a fairly strong association does not necessarily imply that a marker could be successfully used for predicting caries experience. The example they used was the observed strong association between tobacco smoking and lung cancer - the information on smoking status cannot be used to predict the onset of lung cancer accurately, since the majority of smokers never contract the disease. For establishing the value of a potential predictor, methods applicable in the assessment of the accuracy of diagnostic tests are called for. The essence of prediction studies is that they provide us with tools for assessing caries risk in clinical practice, either at the community or individual level, to allow targeted prevention. Specific examples of association include a study of Latvian nursery school children in which no significant associations were found between caries experience and toothbrushing frequency, use

of fluoride dentifrice or parents' education (Bjarnason et al, 1995). Other examples include: a longitudinal study of caries, cariogenic bacteria and diet in children (Holbrook et al, 1995); a longitudinal study of lactobacilli, mutans streptococci and dental caries in children (Roeters et al, 1995) and a study to investigate the relative influence of socio-economic status and behaviour on the dental health of 5 year old children (Schou et al, 1995). Studies which aimed to identify predictors of future caries in children include: a longitudinal study to assess the performance of multiple baseline variables in predicting which children would experience high increments of caries (Bader et al, 1986); the University of North Carolina Caries Risk Assessment Study which included detailed clinical examinations, salivary microbial tests, socio-demographic and dental behaviour data as baseline predictors (Disney et al, 1992); a longitudinal study of caries incidence and caries-related factors by Holbrook et al in 1993; a study aimed to test the predictive ability of defined levels of dietary and oral hygiene habits, mutans streptococci and lactobacilli in saliva in 1.5 year old children (Schroder et al, 1994); and the stepwise prediction of dental caries in children up to 3.5 years of age (Grindefjord et al, 1996).

Stamm et al (1993) outlined a rationale for caries risk assessment. They concluded that an accurate and practical caries prediction model could provide fundamental knowledge about four related goals: targeting those in need; greater effectiveness of preventive procedures; appropriate levels of care and economic efficiency. Many authors continue to strive toward realisation of these goals through caries risk assessment studies. More recently, Tinanoff (1995) noted that although considerable benefit may be achieved when a high risk patient is identified, dentistry has been

slow to adapt to risk assessment and prevention-based practice since this conflicts with the traditions of fee-for-service and procedure-orientated dental education. He added, however, that dental schools are starting to succeed in their efforts to integrate risk assessment and disease management with patient care.

2.2 Literature review

2.2.1 Introduction to literature review

Accepting the multi-factorial nature of the carious process (see Chapter 1), caries risk assessment in pre-school children may be approached using a variety of tools. Many studies have focused on selected associated aetiological factors such as: microbiological factors (Kohler et al, 1988, van Houte, 1993, Roeters et al, 1995, Kreulen, 1997)); diet (Persson et al, 1985, Holm, 1990); previous caries experience (O'Sullivan and Tinanoff, 1993) and oral hygiene habits (Stecksen-Blicks and Holm, 1995). Others have focused on risk markers such as social factors (Gratrix and Holloway, 1994; Schou and Uitenbroek, 1995). Many authors have studied the interactions between a variety of the above risk markers (Bader et al, 1986, Disney et al, 1992, Schroder et al, 1994, Grindefjord et al, 1996, Weinstein et al, 1996, Mattos-Granar et al, 1998). There have been recent challenges to the conventional wisdom that inappropriate bottle use and high levels of oral infection with mutans streptococci are the sole aetiological factors in early childhood caries, hence the shift in terminology from 'baby bottle decay' and ' nursing caries' to early childhood caries (Tinanoff et al, 1997). Some authors have also indicated the influence of indirect factors (Wendt et al, 1995) and a literature review by Eriksen et al in 1991

noted that it is unlikely that the same set of predictors are equally relevant in a global perspective.

It is important to emphasise that the idea of predicting caries is not new (Koch, 1990). However, it is likely that, as yet, we do not have a single highly accurate diagnostic or prognostic test for the detection of the degree of risk an individual has for the development of dental caries (Johnson, 1991). This was also the conclusion from a report of the proceedings of an international symposium, "methods of caries prediction" held in October of 1977 (Bibby and Shern, 1978). More recently, conclusions from an international workshop entitled 'Understanding dental caries' reported that even though a slight improvement in the accuracy had occurred, none of the reported measures for assessing caries risk was accurate enough to be relied on when targeting caries preventive measures (Hausen, 1997). O' Mullane et al (1990) emphasised the need for consideration of practical application of risk models in the field and the effort required in order to allocate subjects to a risk group. Recently, Kawabata et al (1997) noted that attempts to assess risk factors have not progressed to the point of generating information useful in directing resources or in devising interventions.

Following consideration of the statistical approach to caries risk assessment, the following section of the chapter has aimed to provide a review of work carried out on individual factors of caries risk in children followed by a review of studies involving multiple factors. Although the focus has been directed toward studies involving pre-school children, similar work carried out on older cohorts of children using similar methodology and statistical analysis have been considered.

2.2.2 General considerations and methods of analysis

As previously mentioned, caries risk assessment may be approached using a variety of statistical methods. The basic methods of analysis were described in Hausen et al, (1994). They noted that in terms of analysing the data and reporting the results, prediction studies differ sharply from studies with the aim of identifying risk factors that compromise the population's health. In the latter case the evaluation is based on measures of association such as correlation, risk difference, risk ratio or odds ratio. The level of accuracy of a *prediction* regarding future caries development is usually quantified in terms of sensitivity, specificity, positive predictive value and negative predictive value. False positive rate and false negative rate carry exactly the same information as sensitivity and specificity but, in contrast, they reveal proportions of misclassified subjects (Hausen, 1997). Prediction models (or risk models) (Disney et al, 1992) can be created to identify caries risk children, that is those with a dmf > 0. High risk models may be used to identify children at high risk of developing dental caries, that is, those children with a dmf greater than a specified number. This number would be related to the caries prevalence and distribution within a given population (see chapter 4.4.5).

In 1990, Krasse noted that any predictive test must possess at least three characteristics: validity; reliability and feasibility. Validity means that a test must measure what it purports to measure, i.e. high specificity and sensitivity. Reliability in this context is synonymous with reproducibility. When a test is applied to the same subjects on different occasions there should be a high correlation between the two sets of results. Finally, the test must be feasible; i.e. it should be inexpensive

and easy to use. The statistical analyses used in this thesis will be described in chapter 4.4, but a brief outline of the main tests will be described in the following sections.

2.2.2.1 Prediction analysis

2.2.2.1.1 Sensitivity, specificity, positive predictive value and negative predictive value

For establishing the value of a potential predictor, methods applicable in the assessment of the accuracy of diagnostic tests are called for. The level of accuracy is usually quantified in terms of sensitivity, specificity, positive predictive value and negative predictive value. These values are based on cross-classifications of the studied individuals according to their predicted caries risk and the observed actual caries increment during the time period of interest.

In table 2.1, cell 'a' represents the number of subjects with a positive test result (indicating a high predicted risk for caries) whose actual caries increment during the follow-up period was high. Sensitivity (Se) is the proportion of these 'true positives' (TP) among all subjects whose actual caries increment was high. Cell 'd' gives the number of subjects for whom both the predicted caries risk and the actual caries increment was low. Specificity (Sp) is the proportion of these 'true negatives' (TN) among all of those whose actual caries increment was low. Positive predictive value (PV+) is the proportion of 'true positives' among all those for whom the predicted caries risk was high; negative predictive value (PV-) being the proportion of 'true negatives' among the ones whose predicted caries risk was low. The possible range

of these proportions is from zero to one. Often they are also expressed as percentages ranging from zero to 100.

Table 2.1: Measures of the accuracy of a test for caries risk

Predicted caries increment	Actual caries increment		
	High	Low	Total
High	a	b	a+b
Low	c	d	c+d
Total	a+c	b+d	

a = true positives (TP) Sensitivity (Se) = a/a+c

b = false positives (FP) Specificity (Sp) = d/b+d

c = false negatives (FN) Positive predictive value (PV+) = a/a+b

d = true negatives (TN) Negative predictive value (PV-) = d/c+d

A perfect test would have a sensitivity of 100% and a specificity of 100%, implying no errors in risk assessment. Consequently the positive and negative predictive values would be 100%, which means that the predicted high risk group would consist of true high risk individuals only and that only true low risk individuals would be included in the predicted low risk group.

It has been suggested that in a risk model, the sum of sensitivity and specificity should be at least 160% before a caries risk marker can be considered a legitimate

candidate for targeting individualised prevention (Kingman, 1990). This is in agreement with an alternative suggestion by Wilson and Ashley (1989), according to which a sensitivity and specificity of 80% would be acceptable for practical use in the community. If both sensitivity and specificity were 80%, every fifth individual with a true high risk would remain undetected in a risk assessment and thus would not receive the intensified protection against caries that she/he would have needed. Correspondingly, every fifth individual with a true low risk would erroneously be included in the high risk group and receive preventive measures to little or no purpose. Thus, even the proposed minimum acceptable level of accuracy would result in an uninvitingly high rate of misclassification (Hausen, 1997).

2.2.2.1.2 Logistic regression analysis

Regression analysis is a very powerful statistical tool for modelling the appropriate physiological relationship between some numerical response variable and a number of potential predictive factors (Marks, 1990). Regression analysis can model many factors at one time; it can handle not only both numerical and categorical factors, but also model interactions between factors. Logistic regression is a mathematical model formulated to describe a data set and deals with yes / no outcomes (Hall and Round, 1994). In the logistic regression model, the relationship between the outcome and the variables is expressed as a simple equation. The relative importance of each variable is determined by weighting factors or coefficients.

2.2.2.2 Risk Model Development

In the vast field of caries risk assessment, some prediction studies have resulted in the production of risk models (Disney et al, 1990; Marks, 1990; Stewart and Stamm, 1991; Reisine et al, 1994; Grindefjord et al, 1996; Helfenstein et al, 1997, Hausen, 1997). These authors have used varying statistical analyses to create these models but logistic regression analysis is the method most often described. These models have, however, fallen short of the recommended sensitivity and specificity values (Kingman, 1990), which results in a relatively high rate of misclassifications. A large variety of risk markers have been used in the production of these risk models. However, Hausen (1997) noted that the predictive power of even the strongest markers available for review was modest and none accurate enough to be relied on when targeting caries preventive measures. The results of the prediction analysis used for this thesis in terms of targeted prevention and service development will be discussed in chapter 6. Hausen has also reinforced the conclusions of Erikson and Bjertness (1991) who stated that, "as appears from the studies reviewed and from a majority of other caries prediction studies a variety of statistical methods and expressions have been applied to validate results". A direct comparison of total explained variance between the various investigations is, therefore, of limited value. Inter-study comparisons may also be difficult due to different definitions of the criteria for high caries risk and variation in caries prevalence and incidence among different cohorts. In terms of inter-study comparison in the following literature review, a lack of comparability in terms of samples and methodology resulted in few sensitivity and specificity figures being reported.

Recent work carried out on a younger cohort of children by Kawabata et al in 1997 aimed to develop a simple predictive indicator for children of 1.5 years of age. This was based on the environmental living factors affecting the oral health condition of the children at 3-years of age and the resulting Infants Dental Index (IDI) appeared to be valid and applicable for targeting high risk children. The production of this index deviates from the more classical prediction models produced from previous caries risk assessment studies by using a different statistical approach. However, the index was produced from associations between caries onset from 1.5 to 3-years of age and the various factors extrapolated from questionnaire data. The sensitivity for the screening value was 0.56 and specificity 0.57 with PPV and NPV of 0.53 and 0.61, respectively. This indicates that although this method is reasonably effective, it does not meet the suggested sum of 1.6 for sensitivity and specificity before a test can be considered a legitimate candidate for targeting purposes (Kingman, 1990). They did, however, state that although the validity if the IDI was not necessarily high in predicting high risk children, it was considered to be applicable to the field of community dental health in order to educate mothers.

There currently seems to prevail a certain degree of frustration among the researchers who have been doing the best they can to contribute to the development of tools for more accurate caries risk assessments (Hausen, 1997). Many of the limitations have arisen in the statistical analysis and similar techniques have prevailed for many years. One purpose of this thesis was to investigate an alternative statistical approach to caries risk assessment in children to produce an alternative and new model, the Dundee Caries risk Model (DCRM).

The statistical approaches used in this thesis were based on the hypotheses to be tested. The statistical tests used to create the risk models will be fully described in Chapter 4.

For the purposes of this thesis, 'at caries risk' or 'any risk' was defined as any of the children participating in the study who developed caries at 4-years of age, that is a dmf > 0. However, further categories of 'high risk' were developed which were defined as caries at 4-years with a $d_1mft \geq 3$ and a $d_3mft \geq 3$ (see chapter 4.4.5 for the rationale for these definitions).

2.2.3 Dental factors and caries risk assessment

2.2.3.1 Previous caries experience

A relationship between caries experience in childhood and future caries has long been recognised. Tinanoff (1995) noted that the concept that caries prevalence predicts caries incidence (i.e., individuals who have experienced caries are at higher risk for future disease) is well accepted among dental professionals. Birkeland et al (1976), in a study which aimed to compare the caries incidence among children with high and low caries prevalence at the age of 7, noted that there was a significantly different caries activity in these groups from age 7 to 15 and the activity of the groups could be predicted at age 7 years. Children with an initial high caries prevalence had the most marked caries increment. In a review, Hausen et al (1994) noted that past caries experience was probably the most commonly used factor in the assessment of caries risk. In 1986, Bader et al conducted a longitudinal study to assess multiple variables in predicting which children would experience high

increments of caries. Analysis showed that in the 5-year age group, defs was a significant predictor of high caries increment in this group. Greenwell et al (1990), in a longitudinal study, noted that children with pit and fissure decay (primary dentition) were at increased risk of developing subsequent smooth surface caries and children with the faciolingual pattern of decay ('baby bottle tooth decay') were at the highest risk of any group for developing additional carious lesions. This was not a new concept. Hill and co-workers (1967) examined 579 students at ages 6-, 8-, 12- and 14-years to compare caries experience in the deciduous teeth with that in the permanent teeth. They concluded that the children with little caries experience in the deciduous teeth when 6-years of age had the lowest caries increment in the permanent teeth at 8-, 12- and 14-years of age. Other investigators have continued to demonstrate a link between caries in the primary dentition and caries in the permanent dentition. Klein et al (1981) examined the correlation between caries prevalence in the primary dentition at age 7 and the permanent dentition at age 13. They found a significant difference in caries prevalence and increment in the permanent dentition between the children originally having a zero to five deft and those with a deft of six or above. In a longitudinal study of Swedish and Danish children, Poulson and Holm (1980) showed that a statistically significant correlation existed between dental caries in the primary and permanent dentition of the same individual. They did, however, conclude that screening based on dental caries experience in the primary dentition at the age of three seemed to have little practical value in identification of children who later develop caries in the permanent dentition. This result was, therefore, important in terms of the use of this variable in

caries risk assessment. Ter Pelkwijk et al (1990) compared different screening criteria on the basis of caries experience in the deciduous dentition in an attempt to establish a reliable screening method for the prediction of caries. None of the screening criteria resulted in optimal predictive values. They noted, however, that the rationale of using a suboptimal screening test depends on the objective of the screening and a screening test that misses some individuals at risk is acceptable. On the other hand, a screening test that would classify a small number of individuals as false positives is also acceptable since preventive 'treatment' of caries is not harmful for those who actually do not need it. More recently, a study by Raadal and Espelid (1992) aimed to examine the validity of employing the caries experience of the primary dentition for predicting early caries in the permanent first molar fissures. They found that when the children were grouped according to their dmft values, a statistically significant relationship was found between the dmft and number of intact molars in each individual. Results from their analysis may, therefore, be used for selecting children with a specific dmft appropriate for the level of resources available in a fissure sealant program. In a longitudinal study of caries incidence and caries-related factors, Holbrook et al (1993) found that when the data were analysed by stepwise regression, one of the strongest variables was the baseline caries score. Similar significant relationships were found using odds ratios. The presence of caries at 4 years was the strongest single variable associated with a high caries prevalence at 6 years. Recently, Al-Shalan et al (1997) carried out an investigation to determine whether early childhood caries was a risk factor for future dental caries and concluded that indeed a significant relationship existed between

primary incisor caries and future caries in the same individual. They noted that parental education regarding the risk of future caries and preventive counselling should be included in dental planning for these children. A literature review by Demers et al in 1990 stated that past caries experience has been shown as the best indicator of future caries activity among children in studies involving several factors. Other studies investigating risk markers involving older cohorts of children have found a relationship between past caries experience and caries levels (Alaluusa et al, 1990; Mattiasson-Robertson and Twetman, 1993; Holbrook et al, 1995 and Bjarnason and Kohler, 1997).

However, more directly relevant to this thesis has been the work carried out on pre-school children. O' Sullivan and Tinanoff (1993) aimed to quantify the extent of posterior dental caries in those children who initially presented with maxillary anterior caries, and those who did not, in a study of 217 three to five year olds. They found that only 4 of the 38 children with the maxillary anterior pattern did not have a posterior pattern, an indication that once the factors necessary for the initiation of the caries process are established, they are difficult to reverse. They concluded, therefore, that the factors which cause maxillary anterior caries may markedly contribute to the high prevalence of dental caries in the posterior primary teeth. It was also noted that pre-school children need to be the subjects of future caries risk and prevention studies, since the majority of previous studies have focused on older children whose maxillary anterior caries experience cannot be ascertained. In a follow up study of 3- to 5–year old children, Reisine et al (1994) assessed a multidisciplinary caries-prediction model. They noted that, consistent with other

studies of caries risk in children, mutans streptococci levels and previous dmf were important predictors of caries. A high dmf was predictive of future caries incidence. Again, as with the previous study reported, the results implied that once a caries pattern is established, it would be difficult to reverse. They concluded that the results support the argument for the earliest intervention to prevent caries. Early identification of those children at risk, however, is a prerequisite to any early preventive intervention. In the recent study by O'Sullivan and Tinanoff (1996), they also assessed the development of caries in pre-school children according to baseline caries pattern. They showed those 3-year-old children with the maxillary anterior or pit / fissure pattern of caries would have significantly higher levels of caries by the age of 5 than children of the same age who were caries free at baseline. They concluded that in addition to the increased risk to primary posterior teeth contributed by early caries patterns, results from other studies (see above (Greenwell et al (1990), Poulson and Holm (1980)) show that children with posterior caries in the primary dentition may be considered at greatest risk for caries in the permanent dentition. They again stated that efforts to develop caries interventions for children under age three might have a considerable effect in reducing the need for future dental treatment. Grindefjord et al (1996), in a longitudinal study of caries risk in pre-school children, found that children with initial and/or manifest caries at 2.5 years of age were most likely to develop new carious lesion by the age of 3.5-years. The fact that caries prevalence had increased from 11% for initial or manifest caries and 7% for manifest caries to 37% and 29% respectively, indicates that children at risk must be identified early if preventive care can be effective. Past caries

experience is obviously an important predictor, as these authors have noted, however, it has limited use in very young children when very few teeth have erupted.

The results of the caries incidence of the children participating in the present study (see chapter 5.1.1) indicate that establishment of caries occurs prior to age 3-years and, therefore, a comparison can be made to studies by O'Sullivan & Tinanoff (1993) and O'Sullivan & Tinanoff (1996).

2.2.3.2 Parent - child considerations

The studies described in the previous section reported the relationship between past caries experience and future caries increment in the same individual. There have been few investigations, however, relating to the caries incidence of children compared to their parents. Roeters et al (1995) carried out a longitudinal study of 2 – 5.5 year olds to investigate several determinants of caries in this age group. Part of the investigation involved examination of the oral cavity of the parent who was most involved in the education of the child (usually the mother). The level of education and the DMFT score of the mother at baseline were correlated with the $d_b$mft (d_b = loss of enamel surface continuity or dentinal lesions) score of the child. This correlation became stronger with increasing age of the child. The level of education of the mothers showed a higher correlation with the caries experience of the children than the DMFT score of the mothers. Grytten et al (1988), in a longitudinal study of 231 children found that caries experience at the age of 36 months showed a statistically significant association with mother's dental health (as measured by

caries experience of posterior teeth using bitewing radiographs), mother's dental attendance pattern and mother's education. The children of mother's with missing teeth were more likely to develop dental caries. In their caries prediction model, mother's number of missing teeth was the only variable that was significantly associated with caries experience after controlling for the effects of frequency of sugar consumption, mother's education and her dental attendance pattern. They did conclude, however, that none of the variables investigated were strongly enough associated with caries at age 3 to be pointed out as good caries predictors. In a more recent study, Alaluusua and Malmivirat (1994) assessed the ability of chairside tests to identify young children who would experience caries during the subsequent 1.5 year. They found that the DMFT index of the mother was not positively associated with any of the child's caries parameters. Seventy percent of the children whose mothers had high DMFT value were caries free whereas approximately 90% of the children whose mothers had lower values were caries free.

2.2.3.3 Summary of dental factors

To summarise, many authors found that past caries experience was one of the most powerful predictors of future caries in children (Birkeland, 1976, Bader et al, 1986, Greenwell et al, 1990, Demers et al, 1990). A link between caries in the deciduous dentition and caries in the permanent dentition has been well established (Klein et al, 1981, Poulson and Holm, 1980, Raadal and Espelid, 1992, Holbrook et al, 1993). To focus more on pre-school age children studies have found that caries in early infancy may lead to caries in toddlers (O'Sullivan and Tinanoff, 1993, Reisine et al,

1994, O'Sullivan and Tinanoff, 1996, Grindefjord et al, 1996). O'Sullivan and Tinanoff (1993) also noted the importance of caries patterns in the early dentition. Literature on the caries status of the parent with respect to caries status of the child was scant. Roeters et al (1995) found a negative correlation between the DMFT score of the mother at baseline and the dmft score of the child.

2.2.4 Microbiological factors in caries risk assessment

2.2.4.1 Introduction

Hardie (1992) noted that there remain particular groups within the population who still experience high levels of disease and the use of salivary counts of cariogenic bacteria is one way in which such high risk groups could be recognised. Tinanoff (1995) stated that the quantity of specific oral microorganisms is frequently considered to be an important measurable single caries risk factor. In a recent review, Tinanoff and O'Sullivan (1997) stated that it is widely accepted that the group of cariogenic microorganisms mutans streptococci is associated with early childhood caries. They further noted that children with early childhood caries reportedly have elevated oral levels of mutans streptococci which generally are acquired from their mothers. It is well accepted that children harbour many other microorganisms (McCarthy, 1965, Smith, 1993, Pearce, 1995).

A comprehensive review of caries associated microorganisms is outwith the scope of this thesis and the reader is referred to the relevant textbooks and review articles including: Bowden, 1991; Hardie, 1992; van Houte, 1994; Bowden and Edwardsson, 1994 and Bratthall, 1997. The aim of this section of the literature review has been to

concentrate on literature directly related to the involvement of certain micro-organisms in the assessment of the caries risk of pre-school children as monitored by examination of saliva. These organisms include mutans streptococci (*S. mutans* and *S. sobrinus)*, lactobacilli and yeast (*Candida albicans)*. There is no shortage of data with regard to the colonisation by *streptococcus mutans* in the mouths of children (Catalanotto, 1975, Berkowitz, 1975, Bratthalll, 1991 and Grindefjord et al, 1991. These, however, are not directly relevant to the use of microorganisms in risk assessment. An excellent review by Bo Krasse in 1990, stated that although the evidence that mutans streptococci and lactobacilli play a key role for the development of dental caries has grown stronger, no data have been presented which show that a single salivary factor is of significant predictive value. Similarly, Tanzer (1990) stated that there are problems in microbiological monitoring of the oral flora for the purpose of predicting caries. He also noted that to achieve better microbiological predictive data, early carious lesions should be monitored – white spots and chalky areas in fissures - as lesions should be detected as soon after the hypothesised causative events as possible. The further the risk event and the disease detection event are separated by time, the more likely their possible relationship is to have been affected by intervening events of profound significance. It is also recognised that the predominant bacterial flora undergo changes in parallel with the different stages of caries progression (Hardie, 1992), underlying the need for sampling within a certain time of caries diagnosis. Larmas (1992) noted that one diagnostic problem arises from the fact that caries is not a specific infection. There are many people with mutans infection in their oral cavity without any signs of a

caries attack, while abundant carious lesions occur in many patients without mutans infection. Thus the diagnostic value of mutans infection is only relative. It serves to reveal risk factors because mutans is one of the most potent cariogenic microorganisms.

In a review, Beighton (1991) concluded that screening to determine the salivary levels of mutans streptococci and lactobacilli in order to predict the future caries risk of an individual is, at best, likely to identify the low risk group. However, salivary screening as part of a dental preventive and treatment regimen can, without doubt, play an important role. Isokangas et al (1993) stated that results from the literature suggest that tests on mutans streptococci and lactobacilli contribute only marginally and are not cost-effective in the prediction of dental caries if clinical and socio-demographic data are available.

Van Houte (1993) astutely noted that the issue of the suitability of counts of lactobacilli and mutans streptococci for the assessment of caries risk is a complex one. He emphasised that the interpretation of the now significant database is difficult because they involve different subject populations, different methods for caries evaluation and different types of sampling methods. In this review, van Houte stated that according to the literature, prediction of high caries risk in children on the basis of a single microbial factor is problematic. Overall, counts of lactobacilli and mutans streptococci as predictors of caries risk for individuals would appear to be of limited value but the use of such counts for groups of subjects show greater promise. He set out an approach which could optimise the use of microbiological predictors of caries risk including the study of single microbial factors or combinations thereof,

with optimal procedures for sampling, transport of samples, cultural methods and caries evaluation. In a review by Hausen et al (1994) it was noted that the predictive power of mutans streptococci in saliva has not proven better than that of past caries experience. Recently, Bratthall (1997), in an excellent paper, emphasised that those wishing to find more caries in populations highly colonised by mutans streptococci will be disappointed as such simple correlations will never be found world-wide. This, however, does not preclude the use of microbiological data as part of a comprehensive risk assessment study.

2.2.4.2 Methodological considerations

Bratthall and Ericsson (1994) provided a comprehensive review of microbiological tests for the assessment of caries risk including the evaluation of such tests.

In many studies methodological problems associated with the microbiological diagnosis seem to have been treated superficially (Krasse, 1990). He noted that it is taken for granted that the methods and media proved to be valuable in one population and one laboratory also work in another situation. Collaborative studies between different laboratories should, therefore, be encouraged for validation and control of the methods used. The methodology used in the study for this thesis will be fully dealt with in chapter 4. A brief review of the literature regarding methodology is warranted, however.

2.2.4.2.1 Saliva sampling procedure

The selection of the sampling method is dependent on the objective of the examination. (Krasse, 1990). It will also depend primarily upon the age of the population and the method used to cultivate the organisms. Although no comprehensive study has been undertaken to compare the reliability of the various sampling methods, it is very likely that, provided the same one is used consistently within a single study, method will have no important effects on the strength or significance of the associations found (Beighton, 1991). Examples of different sampling methods include: the spatula method (Kohler and Brathall, 1979); the dip-slide system, e.g. the Caries Screen SM (Jordan et al, 1987) and Dentocult – SM (Pienihakkinen and Jokela, 1995); tongue – loop sampling (Beighton, 1986) and pipetting saliva (Alaluusua and Renkonen, 1983). Many authors have compared these types of tests for the recovery of lactobacilli and *streptococcus mutans* (Birkhead et al, 1981; Newbrun et al, 1984; Emilson and Krasse, 1986. Tanzer, (1990) noted that one of the attractive aspects of saliva sampling is the relative mechanical ease of cultivating samples by immersing semi-selective culture medium-coated supports in saliva, or flowing saliva over them, or pressing saliva-wetted devices onto the surface of the medium. These methods, of course, can be no better than the traditional methods of cultivation of diluted saliva volumes on agar plates of the same medium. However, Tanzer (1990) warns that these methods, although widely used because of the simplicity of their collection, samples the teeth as if they were all at equal risk for caries and as if all the surfaces were at equal risk. This says Tanzer, is contrary to fact. He also notes that the time of sampling during

the day profoundly affects the numbers of mutans streptococci and lactobacilli found in saliva, especially if before and after breakfast and toothbrushing. He concludes that it is probable that most studies on risk prediction using microbiological methods have not appreciated this. However, in a previous review, Krasse (1988) noted that in general it is not difficult to determine the approximate level of the number of cariogenic microorganisms of an individual. He referred to the study by Klock and Krasse (1979) in which a significant variation in levels of mutans streptococci and lactobacilli in repeat samples was observed in only 10 per cent of the cases in a study on 655 children aged 9 to 12 years. He also noted that other investigators have found that the numbers of mutans streptococci in saliva samples are fairly stable over short-term periods. Van Houte (1993) also stated that, generally, methods can be a major source of error and that the use of saliva reflects a compromise with respect to test validity, reliability and feasibility. He does, however, recognise that common caries preventive measures involve the whole dentition rather than individual tooth surface areas, obviating the need for precise information of the location of lactobacilli and mutans streptococci within the dentition as provided by plaque samples.

Larmas, in 1992, provided an overview of the caries activity tests for use in everyday dental practice. He noted that the present tests are useful for estimating the caries activity due to bad dietary habits (salivary lactobacilli), establishing the presence of infection (salivary mutans streptococci) and identification of salivary yeasts for the determination of the medical condition of the patient. Davenport et al (1992) evaluated the validity of dip-slide techniques for estimation of salivary

lactobacilli and mutans streptococci levels by comparison with the results obtained from conventional agar plate counts. They conclude that, notwithstanding any minor discrepancies between the two dip-slide methods and conventional counts, the Dentocult tests provide a simple and reasonably reliable means for determining salivary levels of mutans streptococci and lactobacilli. However, determination of caries risk categories should be carried out with caution.

Different methods for sampling yeasts have also been investigated (Berkowitz et al, 1994). These authors compared the use of a modified "tongue blade" technique with one using rinsing / expectorating. They advised that their modified technique would be useful in children and young children.

2.2.4.2.2 Growth and identification of micro-organisms

2.2.4.2.2.1 Mutans streptococci

There is much disagreement about the use of various media for the selective enumeration of mutans streptococci (Tanzer, 1990). The majority of studies have used mitis salivarius agar, as modified by Gold et al (1973). This medium (MSB) relies on a combination of 20% sucrose and 0.2 units per ml of bacitracin for its selectivity for human streptococci. A now extensive literature from several authors indicates that risk assessment studies based on MSB – mutans data are seriously flawed (Tanzer, 1990). The problem focuses on false negative data and highly variable underestimation of the numbers of mutans streptococci. Clearly, concludes Tanzer, there is a need for better, more selective, and less mutans suppressing agars than have been reported to date. However, Beighton (1991) noted that although

other media have been reported as suitable for the selective cultivation of mutans streptococci, none has been widely used except for TYCSB (Trypticase – Yeast – Cystine – medium with sucrose and bacitracin). It was found to be more difficult to enumerate the mutans streptococci on TYCSB because of the growth of many extraneous organisms (Beighton, 1986). Van Houte (1993) again emphasised that the medium of choice for bacterial enumeration is a controversial issue that so far, remains unresolved. It is, however, imperative that bacterial colonies presumed to be different on the basis of their morphology should be adequately characterised by biochemical or serological techniques.

Other methods for identification of mutans streptococci still in their infancy include the use of monoclonal antibodies (de Soet et al, 1987) and genetic fingerprinting techniques (Widerstrom et al 1995). Bratthall (1997) stated that such immunological approaches together with new genetic methods may allow researchers to say who will be heavily colonised by the mutans streptococci and who will not. This has many important future implications in the targeting of preventive care.

2.2.4.2.2.2 Lactobacilli

Lactobacilli can be isolated from saliva using the selective medium described by Rogosa et al (1951), which relies on a low pH and high acetate content for its selectivity.

Recently, Tanzer (1990) observed that there seems little disagreement with the use of Rogosa's SL agar for the selective enumeration of lactobacilli.

2.2.4.2.2.3 Yeasts

The majority of yeasts isolated from the mouth are *Candida albicans* and these can be readily isolated using Sabouraud's dextrose agar, supplemented with 100μg/ml chloramphenicol (Beighton, 1991). Confirmation that the colonies are *C. albicans* can be easily made by determining the production of β - N – acetylglucosaminidase activity.

Another method of yeast isolation reported in the literature uses Pagano Levin agar® containing 100μg / ml 2,3,5 triphenyl tetrazolium chloride and 50μg / ml gentamicin (Berkowitz et al, 1994).

2.2.4.3 Results from cross-sectional studies

This section has focused on cross – sectional studies (studies in which the saliva is sampled only once and the bacteriological results associated with caries experience) in relation to their relevance in the risk assessment of children.

There have been several excellent reviews carried out by Edwardsson (1986); Krasse (1989); Krasse (1990); Tanzer (1990) and Beighton (1991) on this topic but there has been little data on pre-school children. Schroder and Edwardsson (1987) investigated the predictive values of *S. mutans* and lactobacilli in saliva compared to and in combination with defined levels of dietary habits and oral hygiene for the occurrence of caries in 133 3-year-olds. They reported that it was possible to predict caries risk among 3-year-olds and that the addition of bacterial tests enhanced the ability to screen high and low caries risk. The most efficient prediction of high caries risk was achieved when presence/absence of lactobacilli was involved, alone

or in different combinations. Multiple factors in relation to caries will be discussed in 2.2.10. A study by Bretz et al (1992) aimed to seek correlations between *streptococcus mutans* and lactobacilli with the occurrence of decay in 37 3-6-year-old children. Their statistics indicated that the number of surfaces becoming carious increased 1.6 times with an increase of a log unit of mutans streptococci salivary levels. Similarly, the number of surfaces becoming carious increased with increasing levels of lactobacilli and that mutans streptococci and lactobacilli salivary levels were significantly associated with the surface-based caries prevalence rate. They concluded that preventive therapy could, therefore, be directed toward these risk groups with high levels of mutans streptococci and/or lactobacilli. Matee et al (1992), carried out a study which aimed to investigate the prevalence of mutans streptococci and lactobacilli in 34 1 to 2.5-year-old breast-fed children with and without rampant caries. They found that children with rampant caries exhibited statistically significantly higher counts of mutans streptococci in saliva than caries-free children. They did not test the saliva for lactobacilli but found a high prevalence in plaque samples from children with rampant caries. They did, however, note that the use of these microbial parameters in risk assessment is unlikely to be a good predictor of future caries increment since dietary habits change after the termination of either breast- or bottle-feeding. In a wider study, Matee et al (1993) obtained 100 plaque samples from rampant-caries and caries-free children and used biochemical and immunological tests for diagnosis. The isolation frequencies between children with caries and caries-free children were not significantly different. *Streptococcus mutans* was found to be the only streptococcal species and

they concluded that the differences in caries experience could not be explained by differences in mutans species. Reisine and Litt (1993) conducted a study of 481 children aged 3 years to look at markers for predicting caries risk. A saliva sample, questionnaire and dental exam were used to obtain data. Using discriminate function analysis they reported that *streptococcus mutans* has a large and significant effect on caries by itself. They concluded that, consistent with other studies of caries risk in children, their study found *streptococcus mutans* to be the most important predictor of caries experience. They advised that those in lower income groups should receive more intensive involvement in preventive, educational and behaviour modification programmes since they are at greater risk of both *streptococcus mutans* infection and caries development. Again in 1993, Thibodeau et al studied the relationship between salivary mutans streptococci levels and caries in 462 2 to 5.3-year-olds (mean 3.8 years) with similar low socio-economic backgrounds but different ethnicity. Results suggested a direct relationship between mutans streptococci levels and mean dmfs, with increased levels of infection associated with increased dmfs. The strength of this association was greater in Black and Hispanic children than in White children. Therefore, those Black or Hispanic children with high mutans streptococci levels could be categorised as being at highest caries risk. Grindefjord et al, also in 1993, investigated the caries prevalence in 832 2.5-year-old children in relation to a number of variables, including salivary levels of mutans streptococci. Analysis showed that the risk factor (odds ratio) for a child to have caries was 9.3 times higher if the child was colonised with mutans streptococci and 4.6 times higher if colonised with lactobacilli. Mutans streptococci and lactobacilli

were the variables most strongly associated with the occurrence of manifest caries. Boardman et al (1994) published a study of 285 5-year-olds, in widely separated S. African communities, which examined associations between dental caries in primary teeth and salivary mutans streptococci and lactobacilli. Statistically significant and high correlations were found between salivary mutans streptococci counts and dental caries experience in all groups of children but a weak correlation existed between lactobacilli and dental caries. They concluded, therefore, that salivary *streptococcus mutans* counts could offer a useful screening technique for high risk children. Ansai et al (1994), however, suggested that mutans streptococci levels alone are an insufficient indicator for assessing dental caries activity in 4 and 5-year-old children and that the role of lactobacilli and other aciduric bacteria should be considered. Their results from a study of 260 kindergarten children showed a significant but low correlation between the Mucount test (salivary mutans streptococci) and dfs in these children (sensitivity 79% and specificity 44%). This suggested that caries experience is difficult to predict by microbiological variables alone. A comparative study to determine whether lactobacilli or mutans streptococci in saliva better explains the variation of caries in 2728 4-5-year-olds was carried out by Granath et al in 1994. They showed that lactobacilli better explained caries in pre-school children than mutans streptococci, but the higher explanatory value of salivary lactobacilli did not necessarily mean that its predictive power was high. They concluded that mutans streptococci might be more reliable as a predictor, but with comparatively low power. Thus, the question of which of the bacteria is the better predictor is still unanswered. Kohler et al in 1995 again emphasised the

considerable value of monitoring the presence of salivary mutans streptococci at 1 – 2-years of age in selecting children at high risk of developing caries. In a study of 140 3- and 4-year olds they again demonstrated the association between high caries prevalence and high salivary levels of mutans streptococci. In a recent comprehensive study, Zoitopoulos et al (1996) studied the caries prevalence in 641 3- and 4-year olds, of different racial origin, and the salivary levels of caries associated microorganisms (mutans streptococci, lactobacilli and yeasts). They found that the bacteria were isolated at a greater frequency from the saliva samples of the Caucasian children than Afro-Caribbean children in both age groups and yeasts were isolated at a greater frequency from 4-year-old Caucasian than Afro-Caribbean children. The children from whom both mutans streptococci and lactobacilli were isolated exhibited the greatest caries experience, while those from whom neither of these caries associated microorganisms were isolated had the lowest caries score. A study to compare *streptococcus mutans* levels in 7 nursing bottle caries children and nonaffected children within families was carried out by Kreulen et al in 1997. They found that the high caries risk for the nursing bottle caries children in comparison to the non-nursing bottle caries children within families was confirmed by higher counts of *streptococcus mutans*. The correlation between counts and caries risk was, however, not clear between families. They suggested that nursing bottle caries children may acquire cariogenic bacteria earlier and this is in agreement with Kohler et al (1988). One of the conclusions made was that the use of microbiological counts as screening for the assessment of nursing bottle caries does not yield consistent predictive figures. Recently, Mattos-Graner et

al (1998) reported a study of 142 1 to 2.5-year olds that evaluated the relationship between caries prevalence and several factors including salivary levels of mutans streptococci. Significant differences in the mean ds between salivary mutans streptococci levels were observed and children with greater than 50 CFU mutans streptococci had significantly more caries than children with lower levels. A main conclusion was, therefore, that their data confirmed the relationship between mutans streptococci and dental caries in young children observed in different studies. They astutely noted that the prevalence of mutans streptococci (80.3%) was much higher than in many other studies and this may be explained not only by differences in colonisation but could also be influenced by differences in methodologies for sampling and culturing. Roeters et al (1995) also noted that fluctuations of the numbers of mutans streptococci and lactobacilli in samples may be partly due to problems with the sampling technique. Various methodological techniques were described in 2.2.4.2 and it must be emphasised that the studies outlined in this section used many different techniques. However, Krasse (1990) noted that when considering the methodological problems associated with both microbiological and clinical diagnoses, the observed correlations between certain microorganisms and dental caries are surprisingly good. The reason for this must be that both lactobacilli and mutans streptococci play a greater role than other microorganisms in the development of dental caries.

A study of the prevalence of caries and risk factors was carried out on 938 18-month-old children by Weinstein et al in 1996. This used the Cariostat test to measure caries activity (assessment of the acid production of microorganisms).

They concluded that given the low cost and ease of administration and analysis of the Cariostat, it may have some utility in predicting those who will develop caries. It would appear that the caries scoring system used in various studies does not affect the associations found between caries prevalence and *streptococcus mutans* (Koroluk et al, 1995).

Many cross-sectional studies using similar techniques have also been carried out on older cohorts of children. Alaluusua et al (1983), however, noted that since the microflora of children and young children differs considerably from that of the older children, the infection levels of *streptococcus mutans* indicating high caries risk in older children may not be applicable to younger children. Studies, which have found a positive correlation between salivary cariogenic micoorganisms and caries, include: Beighton et al (1987); Wilson and Ashley (1989); Teanpaisan et al (1995); Kohler et al (1995) and Beighton et al (1996).

2.2.4.4 Results from longitudinal studies

Longitudinal studies (studies relating salivary levels of mutans streptococci and lactobacilli to dental caries increments over a defined time interval) will be described in this section. Detailed reviews can be found in Krasse (1988) and Krasse (1990). Two important studies on pre-school children consistently cited in the literature were carried out by Alaluusua and Renkonen (1983) and Kohler et al (1988). The former evaluated the initial establishment, the isolation frequency of *streptococcus mutans* in saliva (and plaque) with reference to caries experience in 45 children from 2 to 4-years of age. They noted that the salivary *streptococcus mutans*

levels were low, and far below the level used to indicate the risk level of older children and that the evaluation of plaque would seem more practical and more reliable than the evaluation of salivary *streptococcus mutans* levels in young children. It should be noted that more recent methodological techniques (Beighton, 1986) have much simplified the collection of saliva from young children. Kohler et al (1988) examined 78 4-year-olds who had earlier been monitored for the presence of mutans streptococci at 4-month intervals from age 15 months to 3 years. Results showed that children colonised at the age of 2 years showed a mean dfs of 5.0, which was significantly higher than the mean dfs in children who were colonised later or were still non-colonised. The earlier the mutans streptococci had been detected, the higher the percentage of children who had caries. Children who were found in the highest class of both mutans streptococci and lactobacilli had ten times higher mean dfs compared with children found in the lowest class for both microorganisms. They concluded that these observations illustrate the value of prevention of early mutans streptococci infection and justify efforts to select at-risk children by microbial means at an early age. A study by Fujiwara et al in 1991 of 356 0 to 2-year-olds evaluated the initial establishment of mutans streptococci and the individual roles of *S. mutans* and *S. sobrinus* in dental caries initiation and progression. Results showed that the detection of mutans streptococci and prevalence of caries increased with age. The number of mutans streptococci determined in the first year significantly correlated with caries prevalence in both years and significant correlations were found between caries increment and the number of mutans streptococci. These results again underline the importance of

these microorganisms when assessing the risk for caries in young children. Reisine et al (1994) assessed the importance of salivary mutans streptococci as part of a multidisciplinary caries-prediction model in 184 children aged 3-5 years. At baseline and 1-year, children were examined for caries and a saliva sample obtained. Analysis showed that mutans streptococci and dmfs in Year 1 were the best caries predictors (see also section 2.2.3.2) in Year 2 and by themselves explained 25% of the variation in decay in Year 2. They noted that, consistent with other studies of caries risk in children, mutans streptococci were found to be an important predictor of caries. More mutans streptococci and higher dmf were predictive of future caries incidence. The results strongly support an argument for the earliest possible intervention to prevent decay – before it develops and establishes a childhood pattern of disease. A more focused study to examine the effectiveness of utilising salivary mutans streptococcus counts in predicting the incidence of dental caries in a 3- to 5-year-old age group (n=148) over a 2 year period was carried out by Thibodeau and O'Sullivan in 1995. They noted that results from the study indicated that a simple direct plate count of mutans streptococci levels in 3-year-old children predicted their future caries risk over a 2-year period. They suggested that children infected by age 3 with high levels of salivary mutans streptococci have high levels of dental caries and are also at greater risk of developing decay than those children who do not harbour the bacteria. They concluded that a truly effective prediction model should identify children at high caries risk before they get the disease and suggested that a practical microbiological assay combined with an appropriate definition of caries risk may improve the ability to predict dental caries in pre-schoolchildren. A

later paper by the authors in 1996 confirmed the importance of salivary mutans streptococcus counts and they concluded that baseline levels may be useful in identifying and predicting caries in pre-school children and that microbiological data may be used to enhance the identification and prediction of caries in the primary dentition, independent of baseline caries status (O'Sullivan and Thibodeau , 1996).

A 3-year study, which related mutans streptococci and lactobacilli to the development of caries, was carried out on a cohort of 252 1.9-2.8-year-old children by Roeters et al in 1995. It was interesting and important to note that high correlations were found between the numbers of mutans streptococci in plaque and saliva samples. Results showed that for the youngest age group, no positive correlation was found between mutans streptococci or lactobacilli and the caries score. Above the age of 3.5 years, however, the correlations with the caries score were higher for lactobacillus counts than for mutans streptococci in saliva. This study did not confirm previous findings that mutans streptococci were isolated more frequently as the child grows older (Fujiwara et al 1991). They concluded that the positive correlations between the numbers of lactobacilli and mutans streptococci in saliva and the caries development in the study were even higher than those reported in several other studies. A further study, which confirmed the association between salivary mutans streptococci and both caries prevalence and incidence, was carried out by Twetman et al in 1996. The 4- to 5-year-old children were clinically assessed at baseline and after 2 years.

A study by Tsubouchi et al (1995), carried out to evaluate the predictive value for caries of the Cariostat test, although on plaque, is important to outline as it examined

100 18 to 36 month-old children. They recognised that it is critical to identify high risk populations using effective screening methods and concluded that the Cariostat test possessed adequate predictive value and should be considered as an effective caries activity test. This was also somewhat reflected in the cross-sectional study by Weinstein et al (1996). Another important study also carried out on plaque by Pienihakkinen and Jokela (1995) determined the practicability of the strip-test in the detection of mutans streptococci infection and its caries predictive value. They emphasised the importance of the early detection of mutans streptococci in children and that screening should be at age 2-years rather than age 3-years.

Other studies, however, question the value of salivary bacterial counts in risk assessment in pre-school children (Alaluusua and Renkonen, 1983, Schroder and Edwardsson, 1987, Holbrook et al 1993, Granath et al 1993, 1994). Again as part of a wider study, Schroder et al (1994) carried out an investigation of 181 children to analyse the predictive ability of mutans streptococci and lactobacilli in saliva in 1.5-year-olds in relation to caries at 3-years. Other variables were also considered. Only 6 children harboured lactobacilli and this variable was omitted from analysis. None of the predictors or combinations of predictors provided high enough sensitivity and specificity values. They concluded that efforts to predict caries development in the primary dentition at an early age were not successful and a large field exists for research on caries prediction in young individuals. A more recent study found that *streptococcus mutans*, among other variables, was not found to be significantly related to the development of caries in 2- to 4-year-olds (Lai et al, 1997). A recent review by van Palenstein Helderman et al (1996) noted that the

differences in caries experience of three continents could not be explained by the prevailing mutans streptococci species. The fact that the cariogenicity of the diet determines the development of dental caries, while hardly affecting the mutans streptococci counts explained the limited value of the latter as an indicator of dental caries. Longitudinal studies, using similar methodologies, carried out on older children which have found salivary bacterial counts to be of use in risk assessment have included: Bader et al, 1986; Crossner and Unell, 1986; Holbrook, 1995 and Vehkalahti et al, 1996. Authors questioning the value of salivary bacterial counts in older children included: Stecksen-Blicks, 1985; Wilson and Ashley, 1989; Alaluusua et al, 1990; Disney et al, 1992; Saemundsson et al, 1992; Mattiasson-Robertson and Twetman, 1993 and Hausen, 1994.

2.2.4.5 Parent – child considerations

One aspect of caries risk assessment of particular interest is the relationship between salivary levels of mutans streptococci in mothers and the subsequent colonisation of their new-born children (Hardie, 1992). The previous sections have described studies which have investigated the role of microorganisms in caries risk assessment in children. Obviously the age and means by which children acquire these organisms is of importance. Bo Krasse (1989) stated that children derive their cariogenic microorganisms from persons in their immediate environment and the mother is in most cases the main source of infection. In 1978, Kohler and Bratthall in a study of 36 4.5-5-year-old children and their parents investigated whether a correlation exists between the number of *streptococcus mutans* in the saliva of

parents and their children. They found that children of mothers with high numbers of *streptococcus mutans* in saliva do not necessarily have the same high level at the age of 5, but there is a definite risk that they will do so. They concluded that there seems to be a low risk for a child to obtain high numbers of *streptococcus mutans* if the mother has less than 100,000 per ml of saliva. These children will probably also have a low caries experience at the age of 5-years. More recent studies in Scandinavia (Kohler et al, 1983, Kohler et al, 1984 and Kohler et al, 1988) have shown that the children of high risk mothers with MS levels of $\geq 10^6$/ml acquire these organisms at an earlier age than those of low risk mothers and, subsequently, develop more carious lesions. More specifically, prima gravida mothers with salivary *S. mutans* greater than 10^6 colony forming units per ml saliva (c.f.u./ml) treated by intensive preventive regimens could delay or prevent the establishment of cariogenic bacteria in their child's mouth and hence dental caries experience for these children was greatly reduced. Aaltonen and Tenovuo (1994) examined the salivary levels of mutans streptococci and lactobacilli in 228 mothers and their children at 7 months of age and five to seven years later in relation to the frequency of maternal salivary contacts. Results showed that maternal salivary lactobacilli were significantly associated with children's dfs/DFS index, but maternal mutans streptococci were not. There were no significant associations between maternal salivary mutans streptococci or lactobacilli levels and the children's levels. The number of decayed teeth in the mothers at the final examination correlated significantly with the children's dfs/DFS score. They concluded that the results suggested an inverse association of frequent mother-child salivary close contacts

with infection by mutans streptococci and incidence of caries in primary dentitions, in a population of first born children whose mothers have high levels of oral mutans streptococci. Li and Caufield (1995) presented the results of a longitudinal study aimed at determining the natural history of the transmission of mutans streptococci from a mother to her child. Using a DNA fingerprinting technique they studied 34 mother-child pairs and monitored the oral bacteria of mothers and their children for approximately 3 years at 3-month intervals. They noted that pivotal to determining the source of mutans streptococci in children is the method for identifying individual strains and showing that the strains found in the mother are the same as those found in her child. The various methodological considerations, however, are outwith the scope of this thesis. Their results suggested that mothers were the major source of mutans streptococci to their children. They were not able to explore in detail the relationship between fidelity of acquisition and caries outcome. Roeters et al, also in 1995, however, although recognising that high correlations between mutans streptococci in parents and children have been reported in other studies, found no positive association between levels of mutans streptococci in the children and their parents in their study. Kreulen et al (1997) in a study described previously in section 2.2.4.3, found that mothers with high levels of *streptococcus mutans* had children with differing levels (even within twins). They concluded that additional factors must, therefore, be involved in the colonisation of children and they hypothesised that the age when the first tooth erupts and the interaction with the child's developing immune system determine the onset of the disease.

In a recent review, Bratthall (1997) described the "Oskarshamm study" in which the salivary counts of every second mother in Oskarshamm with a newborn child were reduced to see if colonisation could be delayed in their children. Even after 15 years, the protected children had less caries compared with the controls – supporting caries as an infectious and transmissible disease. This also supports the suggestion by Hardie (1992) that it would, therefore, be useful to screen mothers at antenatal clinics in the hope of improving the subsequent dental health of their offspring.

2.2.4.6 Summary of microbiological factors

Although it is widely accepted that certain cariogenic microorganisms are associated with early childhood caries (Tinanoff and O'Sullivan 1997), many authors seem unconvinced as regards the use of microbiological counts in the prediction caries (Krasse, 1990; Tanzer, 1990; Larmas, 1992; Isokanges et al, 1993; Van Houte 1993). However, it is accepted that microbiological data can be useful as part of a risk assessment procedure (Beighton, 1991) but the methodological problems associated with microbiological diagnosis should not be treated superficially (Krasse, 1990). Cross-sectional studies carried out which examined the association between microorganisms and caries in pre-school children include Schroder and Edwardsson, 1987; Matee et al, 1992; Reisine and Litt, 1993 and Grindefjord et al, 1993. Many authors support the use of counts of microorganisms in risk assessment (Schroder and Edwardsson, 1987; Bretz et al, 1992; Reisine and Litt, 1993; Thibodeau et al, 1993; Grindefjord et al, 1993; Boardman et al, 1994; Kohler et al, 1995 and Zoitopoulos et al, 1996). There remains some debate, however, regarding

the importance of various microorganisms, namely mutans streptococci and lactobacilli. Other authors have suggested that the use of microbial parameters in risk assessment is unlikely to be useful in caries prediction (Matee et al, 1992, 1993). Ansai et al (1994) suggested that caries experience is difficult to predict by microbiological variables alone and Kreulen et al (1997) concluded that microbiological counts did not yield consistent predictive figures. Longitudinal studies which looked at the predictive capability of certain microorganisms over a period of time include Alaluusua and Renkonen, 1983; Kohler et al, 1988; Reisine et al, 1994; O'Sullivan and Thibodeau, 1996, Weinstein et al, 1996; and Lai et al, 1997. Again, authors found microbial parameters to be an important predictor of caries incidence (Kohler et al, 1988; Fujiwara et al, 1991; Reisine et al, 1994; Thibodeau and O'Sullivan, 1995, 1995; Roeters et al, 1995 and Twetmen et al, 1996. However, many longitudinal studies have not found microbial counts to be predictive of caries incidence (Alaluusua and Renkonen, 1983; Lai et al, 1997 and Van Palenstein Helderman et al, 1996). The relationship between the microflora of parents and their children has also been investigated (Kohler and Bratthall, 1978; Kohler et al, 1988; Aaltonen and Tenovuo, 1994; and Li and Caufield, 1995). There continues to be debate regarding the association between microorganism levels between mothers and their children and subsequent caries progression in the child's dentition.

2.2.5 Dietary factors in caries risk assessment

2.2.5.1 Introduction

A well established link between diet and dental caries has been extensively described in the literature and the reader is referred to relevant texts (Johansson and Birkhed, 1994 and Rugg-Gunn, 1993). A paper by Winter (1988) reviewed the evidence on diet in determining the level of caries risk in the child population. He noted that as far as young children are concerned, the relationship between dietary sucrose and caries has been described as approximating to an S-shaped curve, rising steeply when the sucrose-containing food is eaten frequently, when newly erupted teeth are at risk and when the immune response is immature. He went on to suggest that possibly the age group in which it is best to assess the relationship of dietary factors to caries risk is the pre-school child population. Given that dietary habits formed early in childhood strongly influence eating patterns during the school years (Poulson and Holm, 1980), identification of those at high risk at the earliest possible age would be beneficial. However, as Wendt and Birkhed (1995) noted, longitudinal studies of the influence of dietary factors on caries prevalence in children less than 3-years-old are scarce and a review paper by Demers et al (1990) noted that despite its important role in the aetiology of caries, diet has not been strongly associated with caries. Davenport (1990), also in a review paper on the aetiology of caries in the pre-school child, advised that patterns of sugar consumption, as well differences in the availability of foodstuffs, should be considered. In a recent comprehensive review, Holm (1990) stated that the most potent substrates are the refined carbohydrates and within the multifactorial context,

sugars are the principal cause of dental caries. Much evidence has supported the statement that a high and frequent consumption of sugar by caries susceptible individuals will result in the development of dental caries. The diet of those who constitute the high caries risk group is, therefore, likely to have a high proportion of refined sugars, (Holm, 1990). Holm noted that although many studies have found significant correlations between sugar intake and caries increment, more recent less obvious correlations could be due to the high overall consumption of sugars in industrialised countries. He concluded by stating that the diet of high caries groups is probably very similar whether they live in industrialised or developing countries but what differs is the ability of the individual to resist caries attack. Schou (1991) noted that the use of sugar consumption behaviour as a predictor of future caries experience presents several problems. Firstly, methodological limitations and inadequacies impede the collection of valid and reliable data. Secondly, other important factors that influence caries development also influence the caries-promoting effect of sugar consumption. Persson and Carlgren (1984) noted specific difficulties of dietary studies in childhood. Hausen et al (1994) commented in a review that as a screening criterion for high caries risk, self-reported sucrose intake seems to have little value. This was also the view of Tinanoff (1995) who stated that although sugar consumption was probably the factor most regarded by dental professionals and the general public for assessing dental caries risk, the usefulness of reported sugar consumption in determining caries risk is not impressive. A review by Edgar and Higham (1991) examined diet as a determinant of caries risk and noted that it is not surprising that correlations between dietary sugars and caries are

unspectacular given the underlying difficulties in dissecting out the important dietary predictors of caries. Coupled with low caries increments and the difficulty of statistical handling of the interactions between the collective cariogenicity of the several dietary intakes and their likely individual effects on frequency / caries relationships, it would appear that identification of markers of dietary cariogenicity must be sought from indirect, experimental evidence. Such evidence relating to pre-school children will be described in this chapter. The methodology used for collection of dietary data is provided in chapter 4.3 and its importance as a risk determinant for the study described in this thesis will be discussed in chapter 6.

This thesis focused on dietary factors in relation to the assessment of caries risk in pre-school children.

2.2.5.2 Results from cross-sectional studies

A study by Schroder and Granath in 1983 investigated the predictive value of defined levels of dietary habits as well as oral hygiene in 143 children in connection with the first dental appointment at the age of 3-years. They found that screening children for caries risk by applying different combinations of dietary habits and oral hygiene seemed to be realistic. However, changing oral hygiene habits was more effective for preventing caries than modifying dietary habits. Further analysis by Schroder and Edwardsson in 1987 confirmed the finding that it was possible to predict caries risk among 3-year-olds with the aid of the factors of dietary habits and oral hygiene. Many studies in which diet has been investigated as only one of several factors in the assessment of caries risk have been carried out. Holbrook et al

(1989) produced an index of sugar consumption and found there was a clear threshold of 30 instances of sugar consumption per week above which caries prevalence rose dramatically. Reisine and Litt (1993), in a study of 481 3-and 4-year old children found that baby bottle usage was associated both with higher levels of bacteria, as well as having a direct and significant effect on caries risk. Babies should, therefore, be weaned off night-time bottles by the age of 1-year. Grindefjord et al, also in 1993, investigated caries prevalence in 832 2.5-year-old children in relation to several factors. They found significant differences for meal frequency, candy frequency, and consumption of sugar-containing beverages at night. They also noted that the children with immigrant backgrounds had significantly higher sugar consumption than non-immigrant children and the immigrant children had significantly higher caries prevalence. However, diet was not one of the variables most strongly associated with caries. McMahon et al (1993) studied New Zealand children 2-5 years of age to document their nutrient intake and investigate whether any dietary factor or food pattern could be identified which related to the incidence of dental caries within a fluoridated water supply. No one food group was consumed more by the children with dental decay than caries free children. Children with dental decay did not report eating or drinking more frequently than caries free children and the authors noted that the study provided no evidence that any restriction of intake of sugar-containing foods or beverages would benefit caries status. The Finnish Family Competence Study (Paunio et al 1993) looked at caries at the age of 3-years and found that, when assessed separately, all indicators of sugar consumption were significantly associated with caries distribution. Every second

child with night-time juice had caries and the habit most strongly associated with caries was the use of night-time juice. This was in contrast to a more recent study by Grindefjord et al (1995), which found that juice had no predictive power in determining future caries. A study, which aimed to identify potential indicators of dental caries in primary school children (5-year-olds), was carried out by Gratrix and Holloway in 1994. They found that the high caries group prolonged the use of child feeding bottles and gave fruit juices more regularly to their children. These children were also more likely to be given confectionery regularly when met from school. Stecksen-Blicks and Holm (1995) examined between-meal eating as well as oral hygiene on the dental caries experience of 249 4-year-olds in Sweden. A questionnaire was used to collect information about the frequency of intakes of nine different snack products. Analysis showed that buns and cakes, ice cream, sweet beverages and sweets had the highest correlation to caries experience, but that the impact on caries of toothbrushing frequency was greater than that of snacking. This, however, may have reflected the relatively low validity of the questionnaire when used to assess an individual's between-meal eating. In another multi-variable study, Schou and Uitenbroek (1995) studied the extent to which differences in sweet consumption behaviour, amongst others, resulted in distinguishable differences in dmf by the age of 5-years. Breakfast habits and sweet consumption were statistically significantly related to the caries experience of the children. The more frequently they brushed and the less frequently they had sweets, the less likely they were to have caries. Children without caries experience were more likely to have breakfast at home every day. However, analysis showed that the relationship

between the parents' occupation and the child's dental health was almost four times as large as the relationship between reported sweets consumption and dental health. So, although diet was an important factor, socio-economic status was more powerful in terms of risk assessment. Also in 1995, an important document on the diet and oral health was published in Great Britain - the National Diet and Nutrition Survey: children aged 1.5 to 4.5 years (Hinds and Gregory, 1995, Moynihan and Holt, 1996). The report of the dental survey looked at the drinking and eating practices thought to be related to dental decay. Information was collected by means of an interview. Having a drink in bed every night was associated with having tooth decay among children aged 1.5 to 2.5 and 2.5 to 3.5 years and if the drink consumed in bed contained non-milk extrinsic sugars, the likelihood of experiencing dental decay increased further. The proportion of children with tooth decay among those who consumed confectionery on most days of the week or more often was double that among those who consumed sugar confectionery less frequently. Frequent consumption of carbonated drinks was also related to experience of dental decay. A strong relationship was found between household expenditure on confectionery and dental decay among children aged 1.5 to 4.5-years. It was also found that the benefits of frequent brushing of the teeth did not appear to outweigh the damaging effects of frequent sugar consumption. For example, more decay experience was found among the frequent consumers of sugar confectionery who brushed their teeth more than once per day than among the less frequent consumers who brushed their teeth less than once per day. This survey, therefore, reinforces bad dietary habits as a risk factor of caries in pre-school children. Van Palenstein Helderman et al (1996)

approached dietary factors in children from a different aspect and looked at studies of children from three continents. They found that the differences in caries experience among the three continents could not be explained by the prevailing mutans streptococci species, but instead could be attributed to differences in the cariogenicity of the diets. A study to determine some influencing factors of 'nursing caries' was carried out in 161 2-to-5-year olds by Ayhan in 1996. Their results indicated that one of the effective factors in the development of nursing caries was the bedtime habit. Those children who fell asleep with the bottle had a statistically significantly greater rate of nursing caries than those who did not retain the bottle during sleeping. They also found that the type of carbohydrate liquid did not play an important role in the formation of caries. These results emphasise the importance of mode of delivery of dietary carbohydrate and timing of consumption.

2.2.5.3 Results from longitudinal studies

The many difficulties involved in dissecting out the important dietary predictors of caries have been long recognised (Edgar and Higham, 1991). However, longitudinal studies of markers of caries risk may allow evidence-based prevention toward specific dietary (and other) practices. As previously noted, studies have shown a correlation between high sugar intake and the timing of these intakes. Longitudinal studies focus on the use of such information used in a predictive capacity, that is, can a specific dietary habit predict the future onset of caries in a pre-school child? This section examines these longitudinal studies and the results from the longitudinal study described in this thesis regarding dietary factors will be discussed

in chapter 6. Persson et al, in 1985, compared dietary habits in 261 children at the age of 12 months with the caries status of these children at age 3-years. They found that the children with caries at 3-years had generally consumed cakes, butter, bread and sweet soups more frequently at the age of 1-year. Some "staple" foods (porridge and follow-up formula and meat) were taken more regularly in the non-caries group. The authors concluded that although a feeding pattern at 1-year of age was identified which discriminated between those children who later developed dental caries and those who did not, they do not claim to present a new screening instrument of risk. However, their analysis does indicate that – on the group level at least – a dietary pattern, which may be causally linked with future dental caries development, is already established at 12-months of age. A more recent study again aimed to test the predictive ability of factors at age 1.5-years for caries at 3-years on a population of 181 children (Schroder et al 1994). They noted that no method to date has shown a convincing predictive capacity. Dietary habits were recorded by interviewing the parent with the aid of a frequency form according to Schroder and Granath (1983). They found that unsuitable dietary habits (based on the frequency of intake of cariogenic foods) was a conceivable candidate in a majority of the children. However, the positive predictive value was only 0.26. They concluded that the efforts to predict caries development in the primary dentition at an early age were not very successful and there remains a large field for research on caries prediction in young individuals. Silver (1987) examined 161 children aged 3-years and again at the age of 8-10-years. Dietary habits were recorded at both examinations and social class at the first only. The study showed that a child will

tend to experience around twice as much decay if feeding in infancy was poor as compared to its contemporary in the same social class who was not fed in such a manner and the more favourable the infant feeding practice (when the baby was breast-fed only or no sugar was added to feeds), then the lower was the caries experience. The finding that children given sweetened feeding bottles or comforters in infancy were more likely to be higher sugar consumers at 8-10-years of age supports the idea that the development of a 'sweet tooth' in infancy persists into later childhood. Results from the University of North Carolina Caries Risk Assessment Study (UNCCRA) of grade 1 and grade 5 children showed that diet did not feature as a significant variable. It was almost certain that the one time, one question, self-reporting mechanism was inadequate to elicit the minimum amount and quality of information needed for meaningful analysis (Stamm et al, 1993). This difficulty continues to confront researchers in the field of risk assessment. Again in 1993, Holbrook studied a group of 158 children at age 4-years and again at age 5- and 6-years and measured several caries-related factors including dietary habits. (This author noted that studies on caries prevalence in pre-school children are infrequent probably on account of the difficulty of obtaining a study population - this has been a point raised by almost all authors discussed). Children in the misuse of sugar group had significantly higher caries scores and lower numbers of caries-free children at all ages compared to those who limited sugar intake. He concluded that this longitudinal data served to reinforce an earlier conclusion that the misuse of sugar was one of the strongest factors in determining those children who would develop most caries by 6 years (Holbrook et al, 1993). These results were in

contrast to the more clinical predictors found in the University of North Carolina Caries Risk Assessment Study (Stamm et al, 1993). Using their longitudinal data Holbrook et al (1993) studied several parameters which potentially could be used as a test to predict future caries increment. In this paper, they found that the presence of caries at 4-years was the strongest single variable associated with a high caries prevalence at 6-years. However, when baseline caries prevalence was omitted from the analysis, misuse of sugar was one of the most significant variables and the frequency of sugar consumption as a continuous variable was the most significant factor in the stepwise regression analysis if the misuse of sugar data was omitted. They noted that there are considerable problems in using dietary data in caries risk assessment. However, the questionnaire adopted in the study proved successful in demonstrating a threshold effect of sugar intake. The study reinforces the importance of dietary factors in attempts to assess caries risk in young children. In a similar, more recent study, Holbrook et al (1995) aimed to determine the incidence of caries and the consumption of cariogenic foods between 5-and 6-years of age. Again, they found significantly more caries in those children misusing sugar compared to those who did not. Much of the dietary sugar was taken in the form of soft drinks and the authors noted that the cariogenic potential of this form of sugar consumption should not be underestimated. In 1994, Reisine et al assessed a multi-disciplinary caries-prediction model on a cohort of 184 3-5-year old children. Diet did not appear to be an important variable. However, the children who dropped out of this study seemed to be those at highest risk and this may have affected the prediction results. An excellent paper by Wendt and Birkhed in 1995 described a

prospective, longitudinal study carried out to investigate dietary habits in children and toddlers with special reference to caries prevalence at 2- and 3-years of age. The study showed that differences in dietary habits at the age of 1-year existed between the children who had developed carious lesions at the age of 3-years and children who had not. This was in agreement with the previously described studies by Persson et al (1985) and Grytten et al (1988), who showed that the frequency of sugar consumption at 18-months was significantly related to caries experience at 36 months. Another finding was that neither the frequency of use of a baby feeding bottle nor the habit of giving the child a bottle containing formula at bed-time or during the night seemed to influence the caries prevalence in 3-year-olds. Significantly more children with than without caries at the age of 3 had been breast fed either for a period 2 months or less or for a period longer than 12 months. These findings indicate that it is not the breast feeding per se that causes dental caries but that the breast feeding habit may have an association both with the child's dietary habits and with the rearing practice of the family. It was concluded that screening to identify children with caries risk at 1-year seemed as accurate as screening performed later. Early establishment of suitable dietary habits appeared essential in the achievement of good oral health in children and toddlers. The authors also emphasised the need for further studies on dietary habits and caries prevalence in pre-school children and on the interactive effects of confounding factors. Also in 1995, Grindefjord et al carried out a longitudinal study to evaluate the predictive ability of variables at 1-year of age for caries at 3.5-years of age. Consumption of sugar containing beverages (≥2/day) and consumption of candy (≥1/week) were

among the significant predictors. Again in 1995, Roeters et al described caries prevalence and diet, amongst other determinants of dental caries, during a 3-year observation period of 193 pre-school children. In the 3-year observation period, the children were examined at 6-month intervals until the age of 5-years and the dietary habits recorded. In contrast to the paper by Wendt and Birkhed (1995), these authors found that few children showed other than minimal change in their diet with increasing age. This also contradicted a paper by Rossow et al (1990), in which the frequency of sugar containing products increased from the age of 10 months to 2-years. The authors also found low correlations between the diet and caries scores. They noted that when caries prevalence is low and differences in the dietary habits small, one would expect correlations between diet habits and caries prevalence to be low, and that low correlations may also be partly explained by a low validity of the diet data. Grindefjord et al (1996) furthered their findings in a paper, which described stepwise prediction of caries. They investigated whether risk assessment in two steps during the interval 1-3.5-years of age could improve the predictive ability of identifying those at caries risk before the age of 3.5-years. Among the risk factors at 1-year of age predicting caries at 2.5-years was candy consumption and the risk factors at 2.5-years of age predicting caries at 3.5-years of age included consumption of candy and consumption of sugar-containing beverages. They found that the probability of identifying children at risk for caries development increased longitudinally from 1- to 3.5-years of age and concluded that risk assessment in two steps before the age of 3.5-years would be valuable in targeting high risk children. Their paper emphasised the importance of dietary factors as a consideration in risk

assessment in young children. This is in general agreement with most other authors. A more recent longitudinal study investigated the sequelae of enamel defects in 25, very-low birthweight, pre-term children at 30, 44 and 52 months of age (Lai et al, 1997). They showed that daily sugar intake did not show a significant association with dental caries in these children. However, again as in a previously described study (Stecksen-Blicks and Holm, 1995), caries prevalence was low and this may have affected the result. In 1997, Kawabata et al, in a study of 1575 children initially aged 1.5-years and then followed up at 3-years, aimed to develop a simple predictive indicator for children aged 1.5-years. The items related to caries onset between 1.5- and 3-years of age were cessation of breast-feeding, drinking sweet beverages and current bottle-feeding. They concluded that the indicator (Infants Dental Index) appeared to be valid and dietary factors were amongst the best indicators. However, levels of specificity and sensitivity were not high enough to allow the indicator to be used for prediction but they noted that it was considered applicable in the field of community dental health in order to educate mothers. An evaluation of a new method of selecting risk patients (screening children by a dental assistant using a questionnaire) was carried out on 82 1-, 2- and 3-year-old children in Sweden by Holst et al (1997). The criteria for risk included: more than 6 eatings or drinkings per day; anything but water at night and other oral and clinical variables. Preventive treatment was implemented to those children assessed as dental caries risk. The proportion of children with caries lesions at 4-years (n = 3) and a 'caries risk' assessment at 2-years was 1.0 (sensitivity) The proportion of children with no caries lesions at 4-years (n = 55) and a 'no caries risk' assessment

at age 2-years was 0.70 (specificity). The most frequent risk factors found at age 2-years included frequent intake of sweet drinks. Sweet drinks at night was one of the most common risk factors found at age 3-years. The authors concluded by stating that a risk assessment starting at 1-year makes it possible to predict children at caries risk before manifestation of the caries lesion. They also stated that small children with caries risk can be identified early and the model and strategy used for caries prevention in this study was cost effective and the authors recommended it should be tried and evaluated in other clinics. However, the numbers of children involved in this longitudinal study were small (n = 81 and only 3 children had caries at age 4-years) and this was an interventive, not an observational study as preventive measures were implemented to all those children assessed as caries risk. Therefore, the results should be interpreted with caution and the recommendations may be of limited application.

2.2.5.4 Studies on older cohorts of children

Many studies which have investigated dietary factors and caries prevalence in older cohorts of children have been carried out (Rugg-Gunn et al, 1984; Burt et al, 1988; Wilson and Ashley, 1989). A comprehensive review is outwith the scope of this thesis but some relevant papers will be described. Woodward and Walker (1994) examined data on dental caries amongst 12-year-old children and sugar consumption of the total population for 90 countries. They reported that the all-country data suggest an upward trend of DMFT score with sugar consumption. In industrialised countries alone, the data suggests no relationship between sugar consumption and

DMFT score. However, this by no means negates the fact that sugar can be a factor of importance in caries development, in specific circumstances and in certain individuals. It is possible that it plays less of a role in older children. The authors themselves, however, emphasise the limitations of their data sources. A cross-sectional study by Beighton et al (1996) attempted to elucidate the relations between diet, amongst other variables, and caries prevalence in a group of 328 12-year-old English schoolchildren. They found significant positive associations between DMFT and DMFS and the total number of eating events, the number of sugar-containing eating events and the number of confectionery-eating events, but, overall, no significant associations with the number of starch-eating events. They noted that associations between dietary intake and caries prevalence or incidence are difficult to establish. Again, their findings indicate the importance of frequency of eating, as reported earlier in this section. When the data were subjected to multiple regression analysis, frequency of eating confectionery and sugary foods, but not quantity consumed, was statistically related to caries experience. They concluded by stressing the importance of restriction of the frequency of eating high-sugar foods.

Longitudinal studies looking at dietary factors, amongst others, in older children include work by Wilson and Ashley, 1989; Dummer et al, 1990; and Ekman, 1990. Dummer et al initially looked at 1015 11-12-year olds, followed by 798 of these children at 15-16-years, to highlight factors which had an influence on the caries experience. The amount of money spent on sweets per week was a highly significant factor relating to the caries experience of the children. The relationship between the purchase of sweets and caries prevalence was positive and strong.

Ekman carried out a study which aimed to identify variables that at age 5-years could predict caries prevalence at age 8-years on a cohort of 100 Finnish children. No correlation in frequency of consumption of sucrose-containing products was found. Other variables, such as parents' dental status, were better predictors. More recently, Szpunar et al (1995) assessed the risk from sugar consumption in a population of 429 11-15-year-old children with low caries experience, followed longitudinally for 3 years. They noted that the relation between sugar consumption and caries experience has become less clear in recent years. The associations found between various measures of sugar intake and caries seem to be strong only in populations with a high caries experience. They also state that there have been only a few attempts to quantify the sugar/caries relationship in terms of risk assessment. Their analysis demonstrated a statistically significant positive relationship between sugars as percent of total energy intake and all three forms of the caries variable, and between total sugars intake in grams and total caries increment. They concluded that a higher proportion of total energy intake from sugars increased the probability of caries on all surfaces, and a higher total daily intake of sugars was also associated with total caries increment. Each additional 5g of daily sugars intake was associated with a 1% increase in the probability of developing caries. Those for whom the proportion of total energy intake from sugars was one standard deviation above the mean had 2.0 times the risk of developing approximal caries relative to those one standard deviation below the mean.

2.2.5.5 Summary of dietary factors

It can be noted from the literature described in this section that dietary factors play an important role in both the aetiology of caries in young children but, more specifically, in the assessment of risk for caries. It has been reported that longitudinal studies of the influence of dietary factors on caries prevalence in children less than 3-years old are scarce (Wendt and Birkhed 1995). However, a high and frequent consumption of sugar by caries susceptible individuals will result in the development of caries (Holm 1990). The use of sugar consumption as a predictor of future caries, however, presents several problems (Schou, 1991, Edgar and Higham, 1991). Many cross-sectional studies have found a relationship between sugar consumption and caries prevalence in this age group (Holbrook et al, 1989; Reisine and Litt, 1993; Grindefjord et al, 1993; Paunio et al, 1993; Schou and Uitenbroek, 1995; Hinds and Gregory, 1995). Longitudinal studies have been carried out to investigate the potential for sugar consumption in the prediction of dental caries (Persson et al, 1985; Schroder et al, 1994; Stamm et al, 1993; Holbrook et al, 1993; Reisine et al 1994; Grindefjord et al, 1995). Results have generally been disappointing and most authors have pointed out the difficulties associated with data collection. Schroder et al (1994) concluded that efforts to predict caries development in the primary dentition at an early age have not been successful and there remains a large field for further research. These papers, however, cover a wide spectrum of methodology. Data on dietary habits collected by means of a questionnaire included: Ekman, 1990; Holbrook, 1993; Grindefjord et al, 1993; Reisine et al, 1994; Grindefjord et al, 1995; Stecksen-Blicks and Holm, 1995;

Holbrook, 1995; Grindefjord et al, 1996; Lai et al, 1997 and Kawabata et al, 1997. Interviews were carried out by: Schroder and Granath, 1983; Persson et al, 1985; Reisine and Litt, 1993; Schou and Uitenbroek, 1995; Roeters et al, 1995 and Ayhan, 1996. Another aspect to be considered was the caries prevalence of the population. In areas of low caries prevalence with fewer children at risk, the difficulties of obtaining valid, risk assessment data could be under-estimated. It should be noted, however, that with the decline in caries prevalence in populations, it becomes increasingly important to identify high risk children in order to allow targeted prevention (Demers et al, 1990).

2.2.6 Oral hygiene factors in caries risk assessment

2.2.6.1 Introduction to oral hygiene factors

The factors considered in this section include oral hygiene, in relation to plaque or gingivitis, and toothbrushing, including use of toothpaste. Some degree of toothbrushing would appear to be included as part of the primary socialisation process for the majority of children (Blinkhorn 1978) but its definitive role in risk assessment would seem to remain unclear and the level of use in deprived communities is not well documented. In a paper to review the evidence on oral hygiene in determining the level of risk in the child population, Winter (1988) reported the lack of correlation in many studies between the indices for plaque measurement and caries. He also noted a lack of evidence that mechanical cleaning of teeth, particularly by toothbrushing, is carried out sufficiently well to prevent caries in susceptible individuals. Demers et al (1990) also reviewed oral hygiene

and diet with respect to caries prediction and noted poor association and correlation between these factors and caries. In 1991, a review by Schou noted that caries occurs in the mouth of a person and that person's behaviour and attitudes, as well as the society he or she lives in, inevitably influence not only the occurrence of dental decay but also our possibilities of doing something, be it preventive or predictive. Schou (1991) confirmed that the value of oral hygiene practices against the initiation of caries has been challenged and epidemiological data lacks consistency. One of the reasons for the difficulties in proving the direct relationship between oral cleanliness and dental caries in point prevalence surveys, as well as in longitudinal retrospective or even prospective studies, is the interaction between a number of factors. Tinanoff (1995) noted that toothbrushing has long been a basic component of programs aimed at preventing dental caries, consequently poor oral hygiene is widely considered a caries risk factor. However, he stated that studies have not consistently demonstrated a relationship between dental plaque scores and dental caries prevalence. In a recent review presented at a conference on early childhood caries (ECC), Reisine and Douglass (1998) reported that oral hygiene levels may be associated with caries risk and that increased frequency and better oral hygiene levels are associated with lower caries levels in pre-school children. Again, they noted that a major problem confronting the investigation of the relationship between toothbrushing and ECC is the methodological issue of assessing the frequency of brushing, quality of plaque removal, and actual levels of oral hygiene. Most reports of toothbrushing assess such questions by asking the primary caregiver. These reports are subject to recall bias, as well as to social desirability response bias.

Studies that have assessed plaque scores or gingival status in pre-school children have found a positive and significant association between gingivitis, mutans streptococci and caries (Paunio et al, 1993; Schroder and Edwardsson, 1987). Reisine and Douglass (1998) noted that the data on the relationship between toothbrushing and caries were equivocal and more attention should be directed at the development of more reliable and valid measures of oral hygiene to more accurately assess the effect of this variable on caries risk.

2.2.6.2 Results from cross-sectional studies

Schroder and Granath (1983) studied the predictive value of defined levels of dietary habits and oral hygiene in connection with the first dental appointment at the age of 3-years. Oral hygiene was registered as gingival status. Analysis showed that practising good oral hygiene was more effective for preventing caries than practising good dietary habits. They concluded that children with clean teeth, irrespective of dietary habits, and those with less than one regular unsuitable intake per day, provided they do not have general gingivitis with bleeding on probing, might be regarded as at no caries risk. Children with other combinations of dietary and oral hygiene habits ought to be regarded as at caries risk. Thus, the combination of dietary habits and oral hygiene could be used to predict the risk of caries at the age of 3-years. In a further study, Schroder and Edwardsson (1987), confirmed the prediction of caries risk among 3-year-olds with the aid of dietary habits and oral hygiene expressed as gingival status. A comparison showed that oral hygiene alone was as effective in discriminating low caries risk as it was in combination with

dietary habits. However, the addition of bacterial tests enhanced the ability to screen high and low caries risk. Reisine and Litt (1993) looked at understanding oral hygiene behaviours in 481 3-year-olds. They did not find oral hygiene to be significantly associated with caries risk. In the Finnish Family Competence Study, Paunio et al (1993) examined how dental health habits were associated with dental health at the age of 3-years. They found that daily toothbrushing was significantly associated with low caries frequency. However, the location of caries was not associated with toothbrushing frequency. Dental cleanliness was good in 80% of the children and was significantly associated with low caries occurrence. They also found that in regular fluoride users the proportion of children with caries was significantly smaller than in irregular fluoride users. The use of chewing gum was not significantly associated with caries development. Schou and Uitenbroek (1995) in a study of 520 5-year-olds in Scotland investigated the relative influence of dental health behaviour on the dental health of these children. Toothbrushing frequency was found to be significantly associated with caries experience. They also found that dental health improved with reported toothbrushing habits in each occupational category and that the relationship between socio-economic status and caries experience was considerably more powerful compared with the relationship between toothbrushing or sweet consumption and caries experience. Stecksen-Blicks and Holm (1995) examined the impact of oral hygiene habits on the dental caries experience of 276 4-year-olds in Sweden. They found that the difference in mean dfs between the two groups who brushed once or twice daily and the group who brushed irregularly was statistically significant. Their results showed that the impact

on caries of toothbrushing frequency was greater than that of snacking. This, however, may reflect relatively low validity of the questionnaire used. They noted that the finding that the small group of children who had taken fluoride lozenges had a higher dfs score than the rest of the children probably indicated that lozenges were prescribed when signs of high caries activity were already evident. They concluded that risk factors for dental caries in children are prevalent and early identification of risk should be encouraged and undertaken at an early age. In a study of 631 Latvian 3-4-year-olds, Bjarnason et al (1995) assessed dental health against the background of currently existing conditions. The authors noted that compared with contemporary epidemiological data from other countries, caries levels in Latvian pre-school children were found to be exceptionally high. No statistically significant associations were found between caries experience and toothbrushing frequency and use of a fluoride dentifrice. This was in contrast to the studies previously described in this section. However, the limited use of a fluoride dentifrice could account for the absence of a relationship. The authors also noted that the widely observed social gradient pertaining to oral hygiene habits and caries levels did not emerge in the Latvian population (Grytten et al, 1988; Grindefjord et al, 1993; Schou and Uitenbroek 1995). In a study to determine some influencing factors of ECC, Ayhan (1996) looked at 161 2-5-year-olds with ECC compared with 181 controls. The author found no statistically significant difference between the two groups regarding the age at which toothbrushing commenced and the occurrence of caries. Commenced-toothbrushing age had no effect on caries development. He concluded that the effectiveness of children's toothbrushing is doubtful. This contrasts with

data presented by Paunio et al, 1993) and Stecksen-Blicks et al, 1995, but is in agreement with Roeters et al, 1995 and Bjarnason et al, 1995. Muller (1996) attempted to identify risk-prone children over a 36 month period (11% under 6-years of age had caries diagnosed as nursing bottle syndrome). The author suggested that poor oral hygiene was one of the risk factors for bottle caries and concluded that the risk-prone family should be taught proper hygiene methods. Febres et al (1997) also investigated the relationship between habits and ECC in 100 12-42-month-old children. They found that the distribution of responses to the question "when to start brushing the baby's teeth" was not significantly different between the groups of children with and without ECC. No significant difference was found in the number of times brushing was carried out each day between the groups. As these authors discussed, a factor, which limited conclusions, was the cross-sectional design of the study, as with the other studies described in this section. More recently, Gizani (1998) found that poor oral hygiene and gingival condition were significantly associated with high caries experience in a group of pre-school children. The study consisted of a total of 136 children allocated to one of three groups based on caries experience: the rampant caries group (dmft $\geq$ 6), no caries experience (dmft = 0) and low caries experience (dmft between 1 and 5). Recent results from Scottish epidemiological surveys have shown that the presence of plaque was associated with high levels of decay in 5-year old children (Pitts et al, 1996 and Pitts et al, 1998).

2.2.6.3 Results from longitudinal studies

In a study of 312 children, Persson et al (1985) investigated dietary and dental health information in children at the age of 12-months with the caries status of the child at 3-years. Results showed that children who had their teeth brushed "occasionally or with difficulty" had cavities more often than those whose teeth were brushed more regularly. They noted that less frequent brushing naturally means that the effect of fluoride toothpaste was reduced. Reisine et al (1994) assessed a multidisciplinary caries prediction model in 184 3- and 4-year old children. They showed that brushing frequency was the only significant behavioural predictor of caries change. However, the direction of the relationship indicated that more frequent brushing was associated with *more* decay. A possible explanation may have been that parents overestimated brushing frequency. They noted that parents stated that children were brushing their teeth more frequently in the second year of the study. This is an important point in terms of the limitation of cross-sectional studies on young children, as indicated earlier. The authors emphasised a drop-out rate of 50% and noted that by losing the highest risk children, the explanatory variables may have been less effective in predicting caries. Schroder et al (1994) investigated the predictive ability of defined screening levels of oral hygiene, amongst others, in 181 1.5-year-olds in relation to caries at 3-years. They also considered the use of fluoride in toothpaste and tablets. Oral hygiene was registered as gingival status. Analysis showed that those children who consumed fluoride tablets at 1.5 and 3-years of age differed non-significantly from the group with no consumption at all. With none of the predictors (OH, diet or bacteria) or combinations of predictors was

it possible to find a screening level which combined high sensitivity with high specificity. Only 17 subjects had insufficient oral hygiene and prediction was unaffected by gingival status. They concluded that efforts to predict caries in the primary dentition were not very successful in this study. They contrasted their results with those of Schroder and Granath (1983) and Schroder and Edwardsson (1987) (see section 2.2.6.2), in which higher prediction values were obtained when background factors and caries prevalence were registered at the same age. They also noted shortcomings such as selection of suitable variables and criteria for the prediction of dental caries and the low prevalence of dental caries in this population. Caries prevalence and its determinants were studied in a group of 252 2-to-5.5-year olds by Roeters et al (1995). The children were examined at 6-month intervals in the 3-year observation period. No statistically significant correlations were found between the amount of fluoride ingested or the frequency of toothbrushing and the dmfs score (at the diagnostic level of loss of enamel continuity or dentinal lesions). Between plaque and gingivitis scores and the dmfs score, significant correlations never exceeded 0.21. The authors noted that this was probably the result of the lower caries prevalence and the high level of fluoride ingestion. Grindefjord et al (1995) also evaluated the predictive ability of oral hygiene as a variable in 1-year-olds for caries at 3.5-years of age. They did not find oral hygiene to be a significant predictor in itself, but noted that the high significance of immigrant background (the strongest predictor) probably concealed a behaviour characterised by inadequate standard of oral hygiene, including less frequent use of fluorides (toothpaste and/or tablets), and unsuitable dietary habits which promoted early colonisation by MS and

subsequent caries development. This finding was compatible with the fact that the use of a nursing bottle as well as visible plaque on maxillary incisors in 1.5-year-olds, were significant risk factors for dental caries at 3-years of age (Alaluusua and Malmivirat, 1994). Weinstein et al (1996) also noted that ethnic minority babies face the greatest risk for caries in North America and that a toothbrush and toothpaste may be alien to some ethnic groups who may prefer more traditional oral hygiene practices. In a further paper, Grindefjord et al (1996) investigated variables including oral hygiene at 1- and 2.5-years of age with respect to caries development before 3.5-years of age. Again, they found that children with immigrant background were saddled with a caries risk, although oral hygiene was a directly significant variable. One conclusion made was that risk assessment in two steps before 3.5-years of age would be valuable. Holst et al (1997) assessed 82 1, 2, 3 and 4-year olds for caries risk and provided preventive care. They found that one of the most frequent risk factors found at 2-years was lack of oral hygiene. Visible plaque and the combination of visible plaque and deep fissures in the molars were the most common risk factors found at 3-years. This appears to be in agreement with studies such as Schroder and Granath (1983) and Wendt and Birkhed (1995) who showed that children with good oral hygiene were at lower risk of caries and that good oral hygiene may be able to compensate for unsuitable dietary habits. In a small study of 25 low birth weight children, Lai et al (1997) found no significant association between plaque scores, daily brushing frequency or fluoride exposure and dental caries. However, this was a very small study and there was a low caries prevalence in the study group. Also in 1997, Kawabata et al studied environmental living

factors in children at 1.5-years of age for caries at 3-years of age with the purpose of developing a predictive indicator. Poor oral hygiene was not significantly related to caries onset, but as the authors pointed out, 83% of the children had their teeth brushed by the mother every day. This emphasises the opinions of many authors that good oral hygiene equates to a lower caries risk in children (Schroder and Granath, 1983; Schroder and Edwardsson, 1987; Wendt and Birkhed, 1995).

Fundamental studies carried out on older cohorts of children include the University of North Carolina Caries Risk Assessment Study (UNCCRAS). Disney et al (1992) described the predictors used at baseline used to develop the risk models. The purpose of prediction model development was to identify and select from all available variables those that showed the greatest power in predicting children at high risk as determined by their 3-year time adjusted DMFS increment. Mean plaque score was a significant predictor for grade 5 children in both locations studied but was not one of the most important clinical predictors.

2.2.6.4 Summary of oral hygiene factors

The literature for oral hygiene factors, therefore, appears somewhat inconclusive. In general terms, most authors would appear to agree that oral hygiene is an important contributer to the risk status of a child. Poor oral hygiene has been associated with a higher caries prevalence (Schroder and Granath, 1983; Schroder and Edwardsson, 1987; Paunio et al, 1993; Stecksen-Blicks and Holm, 1995; Muller, 1996) and high caries incidence in terms of prediction (Persson et al, 1985; Disney et al, 1992; Grindefjord et al, 1995, 1996; Holst et al, 1997). Several authors, however, did not

find any significance between oral hygiene and caries (Bjarnason et al, 1995; Ayhan, 1996; Schroder et al, 1994; Kawabata et al, 1997). Many of these studies, however, were carried out in low caries populations.

As stated, oral factors for the purpose of this thesis is defined as oral hygiene and including plaque or gingivitis measurements, toothbrushing and use of toothpaste. Blinkhorn (1978) stated that the definitive role of toothbrushing in risk assessment remains unclear. Winter (1988) reported the lack of correlation between the indices for plaque measurement and caries. Demers et al (1990) have also noted a poor association between oral hygiene and caries with respect to caries prediction. Reisine and Douglass (1998) stated that a major problem confronting the investigation between toothbrushing and ECC is the methodological issue of assessing the frequency of brushing, quality of plaque removal and actual levels of oral hygiene. Cross-sectional studies have found an association between increased levels of oral hygiene and lower caries prevalence (Schroder and Granath, 1983; Schroder and Edwardsson, 1987; Paunio et al, 1993; Schou and Uitenbroek, 1995; Stecksen-Blicks and Holm, 1995; and Grindefjord et al, 1993). However, in contrast, many other authors found no statistical relationship (Reisine and Litt, 1993; Bjarnasson et al, 1995; Roeters et al, 1995; Ayhan, 1996; and Febres 1997). Longitudinal studies, which have shown oral hygiene to be a predictor of caries, include Persson et al, 1985; Disney et al, 1992; and Holst et al, 1997. Others have found oral hygiene to have no significance in terms of caries prediction (Schroder et al, 1994 and Kawabata et al, 1997). The literature, therefore, appears to be somewhat inconclusive but it must be emphasised that many of these studies were

carried out in areas of low caries prevalence with differing methodological techniques.

2.2.7 Social factors in caries risk assessment

2.2.7.1 Introduction to social factors

In a literature review, Schou (1991) stated that social and behavioural aspects of caries prediction were now treated as a separate entity, indicating acceptance that they play a part in the prediction of high risk caries groups and individuals. History has clearly shown a relationship between social characteristics and dental disease patterns and, in particular, how social changes have influenced those patterns. In general terms, social factors are perceived as factors pertaining to the social class structure. However, other socio-demographic factors will be considered in this thesis, such as position in family and mother's marital status. Two main factors are fundamental in understanding the relationship between social status and health (Beal, 1990). The first factor is income, where those in the higher classes in general receive a higher income. The other is education. However, varying classification systems use different indictors. Schou (1991) goes on to point out that social factors are closely linked to behavioural factors, and a great number of behaviours, particularly health behaviours, are characteristic for each social class and thus differ between social classes. Indicators such as: which newspaper the household reads; whether the family has a car or not and number of households with no bath, have been used. However, the potential number of social and behavioural indicators of deprived or disadvantaged groups or individuals is enormous and such indicators

must be carefully chosen before application. Socio-economic status has been recognised for years as one of the main factors influencing equality or rather inequality in general and dental health. Several studies will be described in this section which have used different indicators of social class or socio-economic status (SES). The purpose of this chapter was to review risk assessment in pre-school children, therefore social factors relate mainly to the parents of the children studied, for example: mother's educational status; father's occupation and geographical location. Ethnic origin will also be considered, as several studies have shown significant caries differences between racial groups (Paul and Bradnock, 1986 and Grindefjord et al, 1993). However, Manji and Fejerskov (1994) noted that in studies from the US in which black and white children have been compared in terms of caries experience, only minor differences were found. They emphasised that there are substantial differences in life-style, etc. among people living even within the same area and having, for example, the same income and educational background. Such factors are far more important and there is little evidence for believing in inherent racial differences in terms of innate susceptibility to dental caries. Hunt (1990), in a literature review, emphasised that although a considerable number of studies have shown that various socio-demographic characteristics and selected dental health behaviours are related to increased risk of caries, the potential utility of using information about these additional social risk factors to increase the predictive ability of caries prediction models remains poorly defined. He summarised that most recent studies of caries and SES have identified negative associations. In other words, they have tended to show that caries rates are higher among children of lower

social class for both primary and permanent teeth. Hausen et al (1994) in a review noted that in spite of the clear correlation between social status and caries, in the assessment of caries risk the reported sensitivities and specificities have been low. Further factors that limit the utility of social status as a screening criterion include the fact that social status may affect caries risk differently in different countries, and that screening based on social status may not be considered ethically acceptable. Reisine and Douglass (1998), in a recent literature review noted that two major demographic variables have been addressed in the literature on ECC and caries in the primary dentition. These were 1) race and 2) ethnicity and socio-economic status. They concluded that data suggests increased risk of ECC in ethnic minorities. They also suggest that ethnic minorities may experience significant barriers to dental care, including cost of care and availability of accessible services. Epidemiological studies clearly document the increased risks of ECC associated with ethnic minority status and lower socioeconomic status. Because most studies have been conducted among ethnic minority groups of lower income in the US, it has been difficult to separate the cultural influences of ethnicity from the effects of low SES or poverty status on ECC. Few studies have addressed the joint effects of ethnicity and social class on risk of ECC (Reisine and Douglass 1998). Call (1989) stated that children of low-income families in the United States still remain at significant risk for dental disease. They have higher dental disease rates, higher percentages of unmet dental need and significantly lower utilisation rates for dental care services. A more recent survey carried out in Great Britain (Hinds and Gregory, 1995) showed that children from manual social class backgrounds had

considerably more untreated and treated dental decay than did those from non-manual backgrounds. Experience of dental decay also affected a higher proportion of children whose mothers had no educational qualifications and there was a greater prevalence of decay experience among children from households where the head was unemployed or economically inactive than among those where the head of the household was working. Regarding dental attendance, a greater proportion of children from manual than non-manual home backgrounds had never seen a dentist and children whose mothers had no qualifications were considerably less likely to have been examined by a dentist than those whose mother had a qualification. This report emphasised that social variables indeed play an important part in the differences in decay levels in pre-school children. Schou (1991) also noted that even though many studies have shown significant relationships between social factors and caries experience, few studies have analysed and reported the actual predictive values of these variables. Therefore, following a description of cross-sectional studies which have investigated association-type relationships, studies which have involved the use of social variables to predict caries in pre-school children will be considered.

2.2.7.2 Results from cross-sectional studies

Reisine and Litt (1993) attempted to obtain a better understanding of the mechanism by which social class influences caries risk by focusing attention on an 'at risk' group. The group consisted of 361 3-and 4-year-olds. They noted that the strong effects of class were still evident, even within their relatively homogenised

disadvantaged group. Those with lower incomes, who were unemployed and were non-white had greater risk of being in the caries positive group. They concluded that the consistent and significant effects of social class indicators and ethnicity on oral health status in an already disadvantaged socio-economic group were unexpected and further work is needed to develop better understanding of how class has its effect on the caries process. Also in 1993, Grindefjord et al investigated caries prevalence in 832 2.5-year-olds in relation to social and ethnic background amongst other variables. They found that caries prevalence in children with an immigrant background was significantly higher than in children with non-immigrant background and for those with a low social class compared to families from higher classes. This was in agreement with earlier Swedish studies (Wendt et al, 1991, 1992). Mothers with low levels of education were over-represented in the group of children with caries. The children with immigrant backgrounds had a significantly higher sugar consumption and they also often came from families of lower socio-economic strata. The authors concluded that these factors may explain the higher caries prevalence found in the children with immigrant background. Also, the study indicated that the higher caries prevalence in children of immigrant backgrounds is probably due not only to a dietary pattern predisposing to caries but also to inadequate standard of oral hygiene and low fluoride exposure. In a study of 631 Latvian 3-4-year-old nursery school children, Bjarnason et al (1995) found that caries experience in metropolitan and rural children was virtually identical and independent of sex or ethnic background. They also found no statistically significant associations between caries experience and parent's education. A non-

significant tendency to lower caries levels in children with highly educated parents when compared with the remainder of the sample was observed. The authors indicated that the widely observed social gradient pertaining to oral hygiene habits and caries levels did not emerge in this study and that the results of the study should be extrapolated with care. The randomness of the sample was confounded by children from predominantly well-to-do socio-economic strata, since the relatively high charges for nursery schools are prohibitive to the unemployed and poor. Thus, higher disease levels in children from less privileged groups could have been present outside of the sample. Schou and Wight (1994) studied mother's educational level in relation to their 5-year-old children's caries experience from 324 interviews with mothers of the children who participated in the Scottish Health Boards Dental Epidemiological Programme (SHBDEP 1993/94, Pitts et al, 1994). They found that the mothers' educational level was significantly related to their children's caries experience. Two thirds of the mothers whose children had caries experience at the age of five had finished their education by the time they were 16 years or less, whereas less than half of the mothers with caries-free children finished education at 16 years or less.

Schou and Uitenbroek (1995) studied the extent to which differences in socio-economic status, as measured by the parents' occupation and differences in behaviour (reported previously), already result in distinguishable differences in dental health, measured using the dmf-score, even at the relatively young age of 5-years. They found that no matter which way socio-economic status was measured – either by the mother's education, mother or partner's occupation, which newspaper

they read, how many cars they had or simply by postal code – it was statistically significantly related to the children's caries experience. Children with low socio-economic background were less likely to be caries free. Caries experience increased with both decreasing toothbrushing behaviour and a more manual occupational status. A paper by Provart and Carmichael (1995) showed large differences in the caries prevalence of 5-year-olds from high and low deprivation groups. They went on to report that reductions in caries in these groups with fluoridation were larger for the more deprived, higher caries group. Kinirons and McCabe (1995) investigated the influences on 294 pre-school children's dental health in Northern Ireland with a particular focus on familial and maternal factors. Analysis showed a clear and statistically significant relationship between the mother's level of education and the caries experience of the children. The highest level of education was associated with the lowest prevalence of dental caries. Children who were third and second in order in the family had high proportions who were free of caries, while children who were first born had a low proportion and those who were fourth or more had the lowest. These differences were statistically significant. The authors note that these results were likely due to the first born child being overindulged and increased parenting skills by birth ranks second or third. Children of higher birth rank may receive less attention from the parents concerning preventive behaviours and control of dietary sugars. Roeters et al (1995) investigated social background, as well as other variables previously described, and caries prevalence of 193 2-to-5.5-year-olds over a three-year observational period. Social background was based primarily on the level of education of the mother. Results showed a negative correlation between the

level of education of the mother and the dmfs score of the child which became stronger with increasing age of the child. At every age significant correlations were found between the level of education of the mother and the 1) the daily number of food ingestions and 2) the number of sugar-containing food ingestions of their child. A possible explanation for the correlation between the caries experience in the child and education of the parents, state the authors, could be that more highly educated people demonstrate a more dental minded behaviour: they eat less sweets, brush their teeth more often and visit their dentist more regularly. Muller (1996) attempted to identify risk-prone children and their families through references to the socio-economic status and habits of the family members. The study included 139 children under the age of six who presented for consultation for nursing-bottle syndrome. Muller found that for the risk-prone family: biological parents were usually married; they had an average of 2.66 children and low or moderately low socio-economic status according to ethnic origin. More recently, in a study to investigate the relationship between various social and behavioural factors and baby bottle tooth decay (BBTD), Febres (1997) examined 100 children aged 12 to 42 months and their parents. The children were divided into two groups according to the BBTD status of the child. No significant differences were found between groups for parent's sex, marital status or education, child's type of insurance, and baby's sex. A significant difference was found in the racial distribution of the parents, however, with hispanic parents over-represented among the children with BBTD.

It would appear that the majority of authors found a strong association between caries in the pre-school child and social factors. However, more importantly, can

social factors be used as a predictor of high risk, that is, identify which children will develop caries? Longitudinal studies are required in order to address this question. Work done on older cohorts of children include a study by Dominguez-Rojas et al (1993) who evaluated the possible influence on the development of caries of social class, among other variables, in 1021 students of 6-to-15-years of age. They reported that those belonging to the middle class in comparison to the reference level, low class, proved to have a protection factor which was in accordance with the findings of other authors. The lower classes had more caries, gingivitis and higher plaque scores. Gratrix and Holloway (1994) carried out a study aimed to identify potential indicators of dental caries in primary school children. The study groups consisted of communities whose 5-year-old children had contrasting high (n=144) and low (n=200) caries experience. They hoped that by using both quantitative and qualitative methods hard data might be combined with informed views to reveal new factors previously unconnected with caries risk. The data reinforced the concept that deprivation is associated with increased risk of disease. They concluded that most of the indicators of dental caries in young children identified (rented housing, lone parents, lower social class) were surrogates of, or were associated with, deprivation and poverty, two conditions unlikely to change in the near future. Amstutz and Rozier (1995) examined factors associated with variations in dental caries prevalence using classrooms as a surrogate for the larger community in order to identify community risk indicators (CRI). A total of 6650 students in these sampled classrooms were included in the survey. They found that mean classroom caries scores were lowest in those children with the most highly educated parents. They

noted that although recent studies had shown an inverse relationship between caries prevalence and socio-economic status, for both primary and permanent teeth, in this study only parental education was significant in the multivariate analyses, and only for the Grade K-3 cohort. Holt et al (1996) investigated the relationship between prevalence of disease and social class and ethnic origin in 406 pre-school children in Camden. Although a higher proportion of children in the lower social classes had experienced caries and more had rampant caries, the difference was not statistically significant. In contrast, however, being of Asian origin contributed significantly to the risk of both caries and rampant caries. Jones et al (1997), in an ecological study, investigated the association between dental decay in Scottish schoolchildren and social deprivation as measured by the Carstairs Deprivation score Carstairs and Morris, 1991). They noted that mortality and most diseases show a social class gradient which also exists in the prevalence of dental decay. The four variables used to calculate the Carstairs Deprivation Score include: % of overcrowded households; % of economically active males who are unemployed; % of population in social classes 4 and 5 and % of households with no car. Their results showed a statistically significant and positive correlation in all three age groups of 5-, 12- and 14-years and confirmed that dental decay has a strong positive association with deprivation as measured by the Carstairs index in Scotland at Health Board level. A recent paper by Gibson and Williams (1999) examined the relative significance of dietary sugars, toothbrushing and social class as predictors of caries experience among 1,450 British pre-school children who took part in the National Diet and Nutrition Survey (see earlier, Hinds and Gregory, 1995). Social class was measured by the occupation of

the household head. They found that the prevalence of caries rose from 5% in the youngest group (1.5-2.5-years) to around 32% among 3.5- to 4.5-year-olds. Overall, there was a twofold difference in caries prevalence between the manual and non-manual social classes. They noted that their analysis confirmed the strong relationship of caries with social background. Social class was the most important predictor of caries, after age, and a more powerful predictor of caries experience than the frequency with which the children's teeth were reported to be brushed. They concluded that the findings could imply, that for pre-school children, advice to brush teeth twice a day with a fluoride toothpaste may be more effective in preventing caries than advice to restrict sugars. However, owing to the cross-sectional nature of the study, this hypothesis requires to be tested by other methods.

Studies carried out on older cohorts include Rizk and Christen (1994). In a study of 367 5- to 13-year-olds they found that that children from lower socio-economic families have a trend toward higher caries prevalence rates than children from higher socio-economic families.

2.2.7.3 Results from longitudinal studies

The number of longitudinal studies of social factors in the prediction of caries in pre-school children are few. However, as stated previously, an association of lower social class with caries does not mean this factor can be used in a predictive capacity. A longitudinal study carried out in Sweden (Wendt et al, 1991, 1992, 1999) showed obvious differences in caries prevalence between non-immigrant and immigrant children. They suggested that special preventive dental care programmes

should be developed for immigrant children. In a study to assess a multidisciplinary prediction model, Reisine et al (1994) examined 184 low-income children aged 1-year at baseline and at 3.5-years for dental caries. Demographic factors included: child's age; family size; education and age of parent; family income and race. The authors found that although none of these factors were individually significant, as a block the demographic variables improved the ability of the discriminant function to predict caries. The University of North Carolina Caries Risk Assessment Study (UNCCRAS) as reported by Disney et al (1992) followed over 4000 first and fifth grade children longitudinally for 4-years in a study to develop caries risk assessment models. They noted that important for model application and future research was the lack of association demonstrated for socio-demographic factors. None of the factors including: education of household head; sibling number; age; or sex contributed significantly to any of the four models developed. Race, the one socio-demographic variable that had been statistically significant in the grade 1 Aiken cohort 2-year analysis, failed to meet the statistical criteria for retention in the final 3-year models (see also Stamm et al 1993). They concluded that socio-demographic data contributed little to caries risk prediction over a 3-year follow-up period.

It would appear that, as Hunt (1990) stated, the potential utility of using socio-demographic factors to increase the predictive ability of caries prediction models remains poorly defined. He noted that future research should attempt to address what construct is affected by varying levels of social class or SES, thereby resulting in an altered susceptibility to caries. This search would appear to continue.

2.2.7.4 Summary of social factors

Social factors in this thesis include social class and other factors such as marital status, position in family and ethnic origin. Reisine and Douglass (1998), in a recent review, noted that race and ethnicity and socio-economic status (SES) are two demographic variables which have been recently addressed in the literature on ECC. They concluded that data suggests increased risk of ECC in ethnic minorities, but few studies have addressed the joint effects of low SES or poverty status on ECC. Schou (1991) noted that although many studies have shown significant relationships between social factors and caries experience, few studies have analysed and reported the actual predictive value of these variables. Some cross-sectional studies which have shown a relationship between social factors and caries prevalence include Reisine and Litt, 1993; Grindefjord et al, 1993; Schou and Wight, 1994; Schou and Uitenbroek, 1995; Provart and Carmichael, 1995; Kinirons and McCabe, 1995; Roeters et al, 1995; Muller, 1996; and Febres, 1997. Others did not find a significant association between social factors and caries (Bjarnasson et al, 1995). The number of longitudinal studies using social factors as a predictor of caries in children is few. Many authors did not find social factors individually to be good predictors of decay (Reisine et al, 1994 and Disney et al, 1992). It would appear that, as Hunt (1990) stated, the potential utility of using socio-demographic factors to increase the predictive ability of caries prediction models remains poorly defined. Future research, he noted, should attempt to address what construct is affected by varying levels of social class, thereby resulting in an altered susceptibility to caries.

2.2.8 Medical factors in caries risk assessment

2.2.8.1 Introduction to medical factors

The term 'medical factors' encompasses a multitude of factors including: general medical conditions which may or may not have oral manifestations; physical and medical handicaps and many forms of medication. Schou (1991) outlined some of these factors and stated that from the current literature although it would appear that a mental or physical handicap is not a predictor of high caries risk, nevertheless, handicapped people need special care. She also noted that although progress has been made there is still a group of children at high caries risk caused by the long-term use of sugar-based liquid medicines.

In a review, Winter (1988), cited studies of chronically sick children which showed significant correlations between the use of sucrose-based liquid medicines and caries in the primary dentition (Roberts and Roberts, 1979; Roberts and Roberts, 1981; and Feigal et al, 1984). In a review paper, Shaw and Glenwright (1989) stated that most paediatric medicines are prescribed in liquid form and most have sugar included in the formulation. This has several advantages including masking the taste of the drug, acting as a preservative, antioxidant and as a bulking agent. They emphasised the accumulating evidence of the cariogenicity of sucrose-based medications since the early 1950's. Bentley (1992) noted that an often overlooked source of sugar is that of medicine, which is usually given in addition to other sugar intakes, particularly last thing at night or during the night, when the teeth are most vulnerable due to reduced salivary flow.

2.2.8.2 Studies of medical factors and medication

In a study of forty-four children aged between nine months and six years, Roberts and Roberts (1979, 1981) stated that their results showed that chronic administration of liquid medicines sweetened with sucrose increased the incidence of dental caries in children with chronic medical disorders. No other factors related to diet or dental health practices could be identified to account for the large difference between the study and control groups. Feigal et al (1984) tested the effect of one sucrose containing medication on forty children with chronic exposure to the medicine. They reported a significant difference between patients with a history of liquid medication intake and controls. The University of North Carolina Caries Risk Assessment Study (UNCCRAS) (Disney et al 1992) did not find factors such as: history of ear infection; strep-throat history or antibiotics in last 60 days contributed significantly to the risk models developed. However, they noted that the potential contribution of such health factors is still open to question.

McMahon et al (1993) studied 355 New Zealand children 2-5 years of age and found that 30% had often (more than 6 times per year) received medication. This was usually in the form of antibiotic syrups. In their study, the only factor other than socioeconomic status found to be associated with caries was the relationship with frequent use of medication (sweetened syrups). They noted that this was in agreement with the study by Roberts and Roberts (1979). Paunio et al (1993), as part of the Finnish Family Competence Study, examined how infectious diseases and long-term illnesses were associated with dental health at the age of 3-years in first born children. They noted that children suffer from many infectious diseases during

the first three years of life simultaneously with the eruption of their deciduous teeth and that antibiotics given may affect caries development in two ways. The sweeteners are often fermentable sugars which may increase the risk of caries. Penicillin treatment on the other hand causes a clear, if short lived, fall in the amount of salivary microorganisms such as *streptococcus mutans,* with a possible decreasing effect on caries risk. Their results showed that, in those children recurrently treated with antibiotics, 7% had dentinal caries compared to 9% in children who had received antibiotics less often. The difference was not significant. Long-term illness was present in 14% of the children. 5.6% of children with long-term illness had caries and the authors concluded that parents of children with long-term illness should be encouraged to attend with their child(ren) for dental examinations for early identification of any risks to dental health emerging in the course of the illness.

A study by Gratrix and Holloway (1994) showed that in areas of births of normal weights there was a trend for the low caries areas to have proportionately more births of normal weight than the high caries areas. Similar analysis suggested that the high caries areas had proportionally less uptake of polio vaccination. Reisine et al (1994) did not find antibiotic use to be a significant individual predictor of caries in pre-school children but noted that the demographic variables improved the ability to predict caries as a block. In a recent comprehensive study, Grindefjord et al (1995) did not find chronic illness or chronic disease in the mother to be a significant predictor for caries in children of 3.5-years of age from data collected at 1-year of age. Again, in a follow up study, Grindefjord et al (1996) did not find

either medication or general health to be of significant predictive value at 1 – and 2.5 -years of age with respect to caries development at 3.5-years of age.

Lai et al (1997) studied a cohort of very-low birthweight children (VLBW) and a matched control group (NBW) to determine whether enamel hypoplasia seen in VLBW children predisposed them to increased dental caries risk. Their results showed a strong association of some enamel defects with dental caries in the latter two recall examinations in the VLBW group, but not in the NBW control children. However, the overall susceptibility to dental caries of all the children was low and the VLBW children did not appear to be more predisposed to dental decay than the control children. They concluded that although VLBW preterm children showed a higher prevalence of enamel defects, only one type of severe enamel hypoplasia was strongly associated with dental decay. A risk model for pre-school children was assessed by Holst et al (1997). The model was based on screening of caries risk performed by a dental assistant before the caries attack. Among the criteria for caries risk was: illness for 1 week more than 4 times/year and medication with a saliva inhibiting drug. Frequent illness was one of the most common risk factors found at 3-years of age. Neither of the medical factors was significant at 2-year of age. This, however, was not a prospective study and preventive measures were carried out on the children deemed to be at high risk at age 1-year. Also in 1997, Peretz and Kafka investigated the association between maternal and / or foetal complications during pregnancy and / or delivery and the occurrence of baby bottle tooth decay (BBTD) in the child. Fifty mothers of children with BBTD were used for the study and compared with 50 mothers of children without BBTD (age range 3-

4-years). Results indicated a strong association between the appearance of BBTD and a history of complications during pregnancy and / or at delivery. They concluded that babies born after maternal complications during pregnancy or babies who experience a traumatic birth must be considered to be at risk of developing BBTD when exposed to excessive bottle nursing. The literature, as with so many factors, would appear to show conflicting results. Many authors did not find medical factors to be associated with caries nor have a predictive role. However, these studies were carried out on different populations in different countries and it may be that other factors play a more important role, such as immigrant status. As mentioned previously, this factor may mask other underlying causes and may have done so in relation to medical factors such as untreated conditions.

Holbrook et al (1989) found it encouraging to note that in their study of 4-year-olds, those children who had received a lot of antibiotics in the first 2 years of life, but who took fluoride tablets regularly, had less caries than those children who received antibiotics but not fluoride. They noted that children taking many courses of antibiotics, usually because of chronic ear infection, form a clearly definable group that should be encouraged to use fluoride tablets.

Conversely, dental disease may be used as a risk monitor for medical problems. Miller et al (1986) noted that the presence of dental disease may be an alerting factors to more severe nutritional factors and Dreizen (1989) suggested that the past and present nutritional history of a child might be gleaned from study of the oral structures.

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acid production by microorganisms (Akyuz et al, 1997). A review of these is outwith the scope of this thesis but the author acknowledges their importance. However, one factor, which requires consideration within the constraints of this thesis, is the subjective assessment of caries risk or 'hunch'

2.2.9.2 Studies of other factors

The 'hunch' factor was one of the components of the University of North Carolina Caries Risk Assessment Study (UNCCRAS) (Disney et al, 1992, Stamm et al 1993). As part of the clinical examination to determine the importance of clinical variables in risk assessment the clinician carried out a predicted caries increment score. The predicted caries index reflected the examiner's subjective personal judgement or "gut feeling" about whether a child's 3-year caries increment would be none, low, moderate, or high. By design, no attempt was made to standardise examiners for this subjective index. The results showed that, with only one exception, information from the clinical examination provided the only statistically significant predictors. What may not have been anticipated was the strength of the predicted caries increment score. The importance of predicted caries increment as a risk indicator as measured by its strength and consistency was impressive. The rationale for determining the examiner's subjective judgement of caries risk was twofold. Firstly, the study gave an opportunity to evaluate the possibility that a clinician's global assessment of future caries risk had potential explanatory power that went beyond the hard clinical data being recorded. Secondly, they hypothesised that if certain examiners performed substantially better than others in subjectively predicting

patient caries risk, the opportunity might exist to understand what information was absorbed and utilised by the superior examiners to arrive at the more accurate risk assessment. In this study subjective predictive caries score was a relatively strong predictor of high caries increment in three of the four models. In contrast, the hope that a specific clinical examiner was particularly effective in predicting caries risk was not realised. A study by Isokangas et al (1993) aimed to estimate the clinician's ability to identify, without microbiological or saliva tests, those children who would develop caries within 4 years of the prediction. The predictions were based on the clinical and socio-demographic information routinely available at the annual check-up. The aim was not to explain caries occurrence through a predictive model but to test, in practice, one simple method for the screening of subjects with high caries risk. The results suggested that a clinician could reach the level in the descriptive measures which was generally reached by single or combined saliva tests. The clinicians predicted more than one caries surface for 9.2% of the cases. The true proportion of subjects with caries increment of two or more surfaces was 10.7%. The "best" clinician showed Sn of 79%, Sp of 78%, Npv of 87% and Ppv of 62% in permanent teeth and 67%, 92%, 91% and 67% respectively, in primary teeth. The authors concluded that it would be beneficial if all dentists knew their own ability to identify caries risk subjects. A clinician capable of identifying high risk subjects could be very valuable for a health care centre.

Another aspect of risk assessment of particular importance in this thesis was the involvement of health personnel other than dentists. Mauriello et al (1990) reported on the degree of agreement between dentist-examiners and hygienist-screeners for

specific caries prediction information collected under differing conditions. The study included 5,233 first- and fifth- grade children who were involved in UNCCRAS. Results showed that the DMFT index comparison between hygienist-screeners and dentist-examiners clearly showed that agreement was almost as good as among the dentist-examiners alone (80% vs 85%). The agreement rates for the dmft index between the hygienist-screeners and dentist-examiners were somewhat lower. The remaining indices for which comparisons were made collected descriptive information that may be useful in predicting future caries rates. The dentist-examiner reliability data showed a higher percent agreement and kappa value for three of the indices (morphology, plaque and fluorosis) when compared to the hygienist-screener. Conversely, the hygienist screener reliability data and kappa values were the same or higher than the dentist-examiner for the remaining three indices (referral caries, referral "other" and caries prediction). The authors concluded that for the purposes of determining caries prevalence at the DMFT level, the results of this study suggest that the use of dental hygienists as examiners was a reasonable alternative to the use of dentists. A further paper by Disney et a (1992) using more detailed information compared the effectiveness of caries prediction models using visual / tactile examination data (dentist) with the same models using simplified screening evaluation data (hygienist). They concluded that it was quite clear that screening procedures used by dental auxiliary personnel could achieve a comparable level of caries prediction accuracy with those based on dentist-conducted visual tactile examination. This paper has important cost implications in terms of the use of less costly health care personnel. However, studies to date have

focused on an interaction with dental health care personnel to aid in the detection of high risk children.

This thesis describes the use of health visitors, general health care personnel, in the identification of high caries risk children.

2.2.9.3 Summary of other factors

The other factor investigated in this thesis has been subjective assessment or 'hunch' in relation to caries risk assessment. Many other factors, however, have been implicated in the association with dental caries, such as tooth resistance factors and host immune factors. Disney et al (1992) found that the importance of the predicted caries increment score by the examining dentist in the University of North Carolina Caries Risk Assessment Study (UNCCRAS) was impressive, as measured by its strength and consistency. A similar result was obtained by Isokangas et al (1993), who suggested that it would be beneficial if all dentists knew their own ability to identify caries risk subjects. Other studies have focused on the hunch of health care personnel other than dentists, for example hygienists (Mauriello et al, 1990 and Disney et al, 1992). In this study, health visitors (see chapter 3) were asked to make a subjective assessment for each study child seen. These results will be presented in chapter 5.

2.2.10 Multiple factors in caries risk assessment

2.2.10.1 Introduction to multiple factors

Many of the studies described in this chapter have involved the use of multiple factors for caries risk assessment. The relative importance of the individual factors has been dealt with in separate sections. This section aims to assess the conclusions of authors involved in the risk assessment of children using multiple factors and the relative importance of these factors in caries prediction. Dental caries is a complex chronic disease with a relatively clearly defined aetiology but with a large number of local and general predisposing causes, often with their exact role unclear (Hunter 1988). Hunter then described risk factors for caries under three main headings: diet, microflora and host and added that the whole socio-cultural environment of the community may have an influence on the development of dental caries. The other predisposing factors include: age; socio-economic status (social status, parental employment, parental marital state and maternal health); the level of parental education (especially the mother's ability to speak the indigenous language); and changes in children's diets which may often be influenced by cultural, religious and geographical variations (Davenport 1990). Davenport also recognised that the element of time is required for caries to occur. Krasse (1988), in a review, concluded that the best prediction of future caries activity is obtained by the combinations of factors which are important contributors to the pathogenesis of dental caries. The principal biological factors which have been used as indicators of future caries activity are counts of mutans streptococci and lactobacilli. Winter (1988) concluded that, for the pre-school age group, the collection of data by

parental interview on dietary and oral hygiene practices, the use of sweetened comforters and the prolonged administration of medicinal syrups, together with clinical observations on plaque, gingival status and carious teeth, is likely to yield information which will assist in the identification of high caries risk children. Hunt (1990), in a review, noted that the search for caries risk factors has focused primarily on dental factors such as previous caries experience, or microbiological factors. He suggests that socio-demographic characteristics and selected dental health behaviours are also related to increased risk of caries. Graves et al (1990) focused their review on physical and environmental caries risk factors as studied in the UNCCRAS. Fejerskov et al (1990) stated that attempts to develop simple approaches in the past have evolved from the rather simple approaches, where only one variable has been considered, to those in which a vast array of variables are brought into relationship with the outcome, or response, variable. Demers et al (1990) in review of caries predictors in children noted that studies considering only one factor or a single category of factors at a time did not take into account the multifactorial aetiology of dental caries and previous studies indicated that a single test is not sufficient to predict a high proportion of caries risk children. A combination of several predictors can provide a more efficient screening test than a single indicator. Among them, past caries experience and microbiological factors stand first because they are easy to determine, they show reasonably good association with caries and their combination takes into account the three elements interacting together to produce caries: a susceptible host (past caries experience); a cariogenic microflora (*S. mutans*) and, indirectly, a cariogenic diet (lactobacilli).

The addition of other indicators showing a consistent association with caries, such as socio-economic status, could also increase the predictive power of the model. Depressingly, Fejerskov (1990) noted that it remains a fact that no method of risk assessment has yet been developed that can reliably be used to predict future caries activity, despite considerable efforts having been expended in this direction. Is this because it may be a priori unreasonable to expect that such a model can be developed? Or is it perhaps due to the fact that the analytical tools that have been employed for the development of models are not yet sufficiently refined or do not take into account sufficient number of variables for us to establish a reasonably robust method? Results from more longitudinal caries studies that incorporate multivariate models created prior to designing the controlled studies are needed before a more definite answer to these questions can be obtained. A literature review by Eriksen and Bjertness in 1991 illustrated the limitations linked to the production of multi-factorial prediction models and stated that many of the models or groups of factors presented are conceptual and only partially scientifically tested. They may, therefore, be intellectually stimulating but not very clarifying regarding the relative importance, in relation to oral health status, of the various factors included. Their predictive value is, therefore, limited. These authors also stated that, so far, the combined biomedical and psychosocial approach to oral health had offered more regarding a holistic understanding of dental health problems than establishing a set of ecologically based, efficient criteria for selecting high risk individuals. This literature review found that several predictive tests applied simultaneously give a higher sensitivity than do individual tests alone. Hausen et al

(1994), in a review, noted that the fact that the power of any single predictor has not been satisfactory has led to attempts to improve the accuracy of risk assessment by using screening criteria based on multiple factors. However, in general, the accuracy of multivariate approaches seems to be much lower than one would expect on the basis of the performance of the individual predictors.

A clear message from all these authors is a consistent understanding of the multifactorial nature of the caries process and the importance of an appreciation of this process in terms of risk assessment. Van Houte (1993) noted that the existence of these multiple caries aetiologic factors, as well as their variability and dynamic interaction do not, a priori, bode well for the development of a caries-predictive test which is based on a single parameter. However, Hausen (1997) went on to note that it is this multifactorial aetiolology which makes it likely that even the most sophisticated risk models will be of limited value in predicting future caries development very accurately.

The results of multi-factorial risk assessment studies will now be reviewed.

2.2.10.2 Results of multi-factorial studies of caries risk assessment

Schroder and Granath (1983) in an early study of 143 3-year-olds investigated the predictive capability of defined levels of dietary habits and oral hygiene. They found that oral hygiene turned out to be more effective for preventing caries than dietary habits and that a combination of dietary and oral hygiene habits could be used to predict the risk of caries at age 3-years. A further study by Schroder and Edwardsson (1987) aimed to investigate whether dietary habits, oral hygiene and

presence / absence of *S. mutans* and lactobacilli, singly or in combinations, could be used as caries predictors for 133 3-year-olds. They concluded that the addition of bacterial tests as predictors, alone or in combination with dietary and oral hygiene habits, enhanced the ability to screen high and low caries risk. The most efficient prediction of high caries risk was achieved when the presence/absence of lactobacilli was involved, alone or in different combinations. Again, they found that practising good oral hygiene was a more effective caries preventive measure than satisfactory dietary habits. In these two studies, the independent variables were registered retrospectively on the same occasion as the caries examination. There is, therefore, a need to test the prospective predictive power of relevant variables at an earlier age, so that intensified preventive advice can be given to parents of identified risk individuals. A more recent study by Schroder et al (1994) involved a longitudinal study of 181 children to test the predictive ability of several variables at age 1.5 for caries at age 3 years. The test variables included: general health and medication; fluoride supplementation; dietary habits; *S. mutans* and lactobacilli counts; clinical examination; oral hygiene. Analysis showed that with none of the predictors or combinations of predictors was it possible to find a screening level which combined high sensitivity with high specificity. The authors noted that besides the difficulty in selecting suitable variables and criteria for the prediction of dental caries, other shortcomings such as the low caries prevalence should be born in mind.

Ekman (1990) carried out a longitudinal study of 100, 5-year-old Finnish immigrant children to identify variables at age 5 that could predict caries prevalence at age 8-years. The author observed the development of caries, dietary factors and parents'

attitudes and attendance. In the first analysis, using caries experience at the age of 5 as the predictor, 73% of the children were correctly classified. Using the parent's dental status as the predictor, 84% were correctly classified according to their DFS score. It was noted that although it would seem relevant to attempt to predict caries by using caries experience as a predictor, this method has had discouraging results in the past (see also section 2.2.3.2). In conclusion, screening at the age of 5-years, based on the parent's dental status could have some practical use in this immigrant group. It must be emphasised that this study focused on an immigrant group in Finland and these methods may not be applicable to other groups. One of the best documented longitudinal risk assessment studies was the UNCCRAS (Beck et al, 1992; Disney et al, 1992; Graves et al, 1992; and Stamm et al 1993). This study was conducted on 5,233 grade 1 and grade 5 children in low fluoride communities surrounding Aiken, South Carolina and Portland, Maine. Although these studies were carried out on young schoolchildren, not pre-school children, results and methodology were fundamental to the field of risk assessment. The information obtained was divided into four categories. Firstly, clinical variables which included: caries diagnosis (D_1 threshold); oral hygiene (plaque index); predicted caries increment (hunch) and pit and fissure morphology. Secondly, microbiological variables which included salivary mutans streptococci and lactobacilli. Thirdly, socio-demographic data which included social, demographic, family and personal history data. The fourth category, health behaviours, included: frequency of between meal snacks; fluoride use; toothbrushing frequency and mouthrinses. A total of 47 independent variables were included. Analysis showed that the logistic

regression caries risk prediction model for the Aiken grade 1 cohort included only four statistically significant predictors. These were baseline DMFS and dmfs, pit fissure morphology score and the clinician's subjectively determined predicted caries increment. For the Aiken grade 5 cohort, six predictor variables achieved statistical significance: DMFS, morphology score; number of sound permanent tooth surfaces at risk; mean plaque score; dentist's referral score (urgency of the child's need for caries treatment) and lactobacillus score. For Portland, significant predictors for grade 1 were pit and fissure morphology and the clinician's prediction of caries increment. For grade five significant predictors included DMFS, sound permanent tooth surfaces at risk, mean plaque index and clinician's prediction of likely caries increment. These were 'high risk' models as they had a defined level of caries increment. The 'any risk' models (DMF > 0) were similar. Some additional variables added when the models became 'any risk' included: mutans streptococci score; race; toothbrushing habit; education of household head. These were for the Aiken models. The comparability between these two modelling approaches appeared to be maintained for the two Portland grade cohorts. A result of major interest was the predominance of clinical measures from the intra-oral examination as significant predictors of future high caries increment occurrence. The significance of the pit and fissure morphology score and the clinician's subjective prediction of the future three-year caries increment (see section 2.2.9) were surprising for both their strength and relative consistency across the models. The authors noted that the relative weakness of the microbiological variables to predict high caries increment in this study was disappointing (see also section 2.2.4), as a

great deal of expense and commitment had gone into this. The ineffectiveness of socio-demographic and dental behavioural variables to predict high caries risk may have been due to the limit of large scale field studies to accurately determine or measure the type of information being sought. Stamm et al (1993) outlined the major conclusions of the study and noted that it obtained three-year predictive specificity values of nearly 0.83 and a caries prediction sensitivity of approximately 0.60. Although clinical variables were the most predictive, Stamm suggested that more refined methods than those employed in large scale epidemiological field studies must be employed to search for aetiological relationships that almost certainly exist.

Another fundamental longitudinal prediction study was carried out in Sweden on pre-school children (Grindefjord et al, 1991; Grindefjord et al, 1993; Grindefjord et al, 1995; and Grindefjord et al, 1996). A group of 692 children were followed longitudinally from age 1-year to 3.5-years. The authors assessed variables associated with caries which included: *streptococcus mutans* establishment; dental factors, dietary factors; social factors, such as mother's education and immigrant status; oral hygiene and dental behaviours. Early results implied an increased risk for caries in 1-year-olds with early colonisation with mutans streptococci (Grindefjord et al, 1991). In a further cross-sectional analysis, the variables most strongly associated with caries at age 2.5-years were colonisation with mutans streptococci, lactobacilli and immigrant background (Grindefjord et al, 1993). It is important to note that the children with immigrant backgrounds had a significantly higher sugar consumption and also came from families of lower socio-economic

status. It is possible, therefore, that immigrant status may mask a host of other factors important in prediction (see also section 2.2.7). In a further paper, the authors aimed at evaluating the ability of dietary habits, oral hygiene factors, fluoride exposure, occurrence of mutans streptococci and social and immigrant background in 1-year-olds to predict caries before 3.5-years (Grindefjord et al, 1995). Analysis showed that of the variables studied, immigrant background exhibited the highest sensitivity (0.77) but a low positive predictive value (0.43). Mutans streptococci was the variable with the highest predictive value for a positive test (0.61), but the sensitivity was rather low (0.13). The authors reported that the results showed that socio-demographic factors, dietary habits and occurrence of mutans streptococci were significant predictors to early caries development. Of the socio-demographic variables studied, mother's education was a significant predictor to caries development. They concluded that the results strongly indicated that risk assessment of children at 1-year of age should be performed, based on the assumption that an individual preventive programme has to be created with respect to the specific profile of predictors. Further study of these children (Grindefjord et al, 1995) followed the progression and development of caries lesions in the children from 2.5- to 3.5-years of age. The caries prevalence increased from 11.3 to 36.7% in this 1-year interval. Ninety-two percent of the children with caries at baseline developed new carious lesions during the following year and 64% of the lesions diagnosed at baseline as initial caries had progressed to manifest lesions at follow up. The authors concluded that children with (chronologically) early caries development exhibited high caries progression and also continued to develop

extensive numbers of new lesions during a 1-year period. Consequently, this group of children need early and improved preventive care to avoid extensive destruction of the primary dentition. More recently, the authors investigated the caries predictive ability of the afore-mentioned variables in children at 1- and 2.5-years of age with respect to caries development before the age of 3.5-year – so called stepwise prediction. The risk factors at 1-year of age, which predicted caries at 2.5-years, were: mutans streptococci; immigrant background and consumption of candy. The risk factors at 2.5-years of age, which predicted caries at 3.5-years, were: mutans streptococci, mother's education; immigrant background; consumption of candy and consumption of sugar-containing beverages. The authors stated that the probability of identifying children at risk of caries development increases longitudinally from 1- to 3.5-years of age, as does the number of predictors. Furthermore, the effect of each predictor increased significantly between 2.5- and 3.5-years of age compared to the interval 1- to 2.5-years of age, with respect to caries incidence. If only immigrant children to mothers with low levels of education were considered for the mutans test, 32 out of 173 children could be detected with a sensitivity of 0.74 and specificity of 0.63. It was, therefore, concluded that risk assessment in two steps before the age of 3.5-years would be valuable in targeting children at high risk for early caries development. The risk profile of each child should be established at 1-year of age based on information on socio-economic and immigrant factors, sugar consumption and oral hygiene habits. Immigrant background and high sugar consumption should be considered as the most significant risk factors. Based on the fact that the caries incidence increased

significantly between 2.5- and 3.5-years of age, it should be reasonable to suggest a new risk assessment and a dental examination by the age of 2.5-years. At this age a test for mutans streptococci should improve the possibility to identify children most at risk.

The differences between the results of the UNCCRAS and this Swedish study could be of geographical origin. Immigrant status was identified as one of the strongest predictors of caries risk in the Swedish study. The cohort used for UNCCRAS did not have this high proportion of immigrants and, therefore, other factors emerged which were significant predictors, such as clinical data. No subjective assessment was made by Grindefjord and co-workers and clinical factors were not as strong. It is important to note the differences in the results of the microbiological factors. These methods are costly and time consuming and this must be taken into consideration when combinations of other factors could be used for successful prediction (see also section 2.2.4).

Another longitudinal study carried out by Holbrook et al (1993, 1995) analysed caries prevalence, caries incidence, factors associated with caries and the prediction of caries incidence. Variables measured included salivary mutans streptococci and lactobacilli, salivary factors, diet and dental health behaviours. The children were initially aged 4-years and followed longitudinally for 2 years. Holbrook (1993) found strong associations between high counts of *streptococcus mutans* or lactobacilli and caries and the misuse of sugar was strongly associated with a high caries score. In terms of prediction (Holbrook et al, 1993), the presence of caries at 4-years was the strongest single variable associated with a high caries prevalence at

6-years. Once this was omitted from regression analysis, the significant variables were misuse of sugar, salivary pH, and counts of mutans streptococci and lactobacilli. They noted that the combination of several variables improved their individual predictive power and that although past caries experience was a strong factor associated with further caries, it is of limited value as a predictor in very young children when caries prevalence is very low. They concluded that a multifactorial model test for caries activity would be more reliable than any single factor and that high counts of mutans streptococci or lactobacilli or the misuse of sugar or regular use of sugar-containing paediatric medicines were regarded as caries susceptibility factors. A smaller study of 43 of these children before and after starting school (Holbrook et al 1995) was carried out to help explain the continued high caries levels. Results showed that much of the new caries was seen in those children who already had caries at 5-years of age. Significantly less caries was observed in the children who did not carry *streptococcus mutans* in their mouths. There was a significant association between sugar intake and caries score. The authors concluded that the increase in sugar intake suggested a change in habits concurrent with commencement of school, thus there was a need for a far greater emphasis on caries prevention through dietary measures in this population. These studies were interesting although they were carried out on an older cohort of children than the present study (4-6-years). Dietary factors were very significant in both association with and prediction of caries, as were microbiological factors. Again, comparison with the Swedish study and UNCCRAS showed differences in the predictor variables. The high significance of dietary and microbiological

variables was not seen in UNCCRAS. Holbrook and co-workers did not measure the important variable of clinician's subjective assessment, nor other clinical variables. Therefore, other variables assume greater importance.

Other studies, which have investigated multiple factors in caries risk, include that reported by Reisine and Litt (1993). They studied 481 3-year olds in relation to dental health behaviour, diet, salivary *S. mutans* and psychosocial factors. They found that race was consistently an important predictor of caries in all the multi-variate analyses. They also found that *S. mutans* was the most important predictor of caries experience and that baby bottle usage was associated both with higher levels of bacteria, as well as having a direct and significant effect on caries risk. The effects of social class indicators and ethnicity on oral health were significant. A surprising finding was that children of parents with higher stress levels had fewer carious lesions than those with low stress. This may have been due to the measure used but requires further research. The authors concluded that those in lower income groups should receive more intensive involvement in preventive, educational and behaviour modification programmes since they were at greater risk of both *S. mutans* infection and caries development. In a further study, Reisine et al (1994) assessed a multi-disciplinary caries prediction model. 184 3-5-year-old children were examined at baseline and at 1-year later for dental caries. Variables examined included: socio-demographic factors; medicines; dental health behaviours; salivary *S. mutans* and psychosocial factors. Analysis showed that mutans and dmfs at baseline were the best caries predictors in for caries a year later. None of the demographic individual variables were significant but as a block improved the

ability of the discriminant analysis to predict caries. An important point to note was that those who dropped out of the sample seemed to have more caries and caregivers with relatively lower educational levels. They concluded that the most important predictors of future decay were dmfs in the previous year and *S. mutans* levels and that future work should address the potential of structural modelling to analyse the direct and indirect effects of cognitive factors on caries risk.

Paunio et al (1993) examined how dental health habits affected dental health in 3-year-old children and whether illness and its treatment were associated with the occurrence of caries. They found that when assessed separately all indicators of sugar consumption were significantly associated with caries distribution. Again, when assessed separately, toothbrushing and use of fluoride tablets were associated with caries distribution. The habit most strongly associated with caries was the use of night-time juice. They concluded that differences in dental health between 3-year-old children could be explained by dental health habits. These habits support the idea that the frequency of sugar intake could be of importance. However, this was not a longitudinal study and no predictive capabilities were given.

Schou and Uitenbroek (1995) studied the extent to which differences in socio-economic status and dental health behaviour already result in distinguishable differences in dental health at age 5-years. The children whose mothers were not interviewed had more decayed teeth than those whose mothers were interviewed. As mentioned previously, this is important in terms of interpretation of the results. The study demonstrated a significant relationship between mother's and children's behaviour, as well as socio-economic status, and the children's caries experience.

No microbiological variables were investigated. Kawabata et al (1997) aimed to develop a simple predictive indicator for children of 1.5-years of age based on environmental living factors and caries incidence of the same children up to 3-years of age. The factors related to caries onset between 1.5- and 3-years of age were cessation of breast-feeding, drinking sweet beverages, watching TV during meals and current bottle feeding, in order of the highest partial correlation coefficient. Sensitivity of the model was 0.56, specificity 0.57. The authors concluded that the Infant's dental index (IDI) developed in the study appeared to be valid, hence it could be applied in the field of community dental health to identify higher risk children and direct more effective health education to mothers. It must be noted that these sensitivity and specificity values do not meet the levels considered to be legitimate for targeting individualised prevention (Kingman 1990) and no microbiological factors were considered. Ollila et al (1998) carried out a longitudinal study to assess the progression of caries with particular reference to aetiological factors. They found a borderline association between early colonisation of oral lactobacilli and candida and an increased risk of developing caries in young children. Pacifier-sucking and the use of a nursing bottle at night were risk factors for the colonisation of oral lactobacilli and candida. These variables were also found to be strong risk factors for caries development. If the sucking habit lasted less than two years it was not so harmful. They did not observe an association between prolonged breast-feeding and caries development. This is in agreement with Weerheijm et al (1998) who showed that prolonged breast-feeding on demand did not lead to a higher caries prevalence. Ollila et al (1998) concluded that although

there was not necessarily a cause and effect relationship between these two factors and caries, prolonged use of a pacifier and a nursing bottle at night were possible risk factors for caries in children. Also in 1998, Mattos-Graner et al evaluated the relationship between caries prevalence and a number of factors including clinical, microbiological and dietary factors in 142 1- to 2.5-year-olds. Significantly higher prevalence of manifest caries was observed in children who were bottle-fed with milk, sucrose and cereals when compared with other groups (milk with or without sucrose). This suggested that the combination of sucrose and starch could be more cariogenic than sucrose alone. Children who were never breast-fed or breast-fed only until 3-months of age exhibited a higher caries prevalence than children breast-fed for a longer time. A significantly higher caries prevalence was observed in children with visible plaque. Children with high salivary levels of mutans streptococci had a mean ds significantly higher than children with 0 CFU or 1-50 CFU. They noted that their study confirmed the relationship between caries in young children and mutans streptococci seen in other studies (Grindefjord et al, 1993, see also section 2.4). They concluded that the variables most related to dental caries in 1.0 to 2.5-year olds were salivary mutans streptococci levels and plaque accumulation on maxillary incisors. Dietary variables could also be related to caries to a lesser extent. Gibson and Williams (1999) examined associations between social class, toothbrushing habit and dietary factors with dental caries in pre-school children. The children studied participated in the National Diet and Nutrition Survey of Great Britain (Hinds and Gregory, 1995). Analysis confirmed the strong relationship of caries with social background. Social class was the most powerful

predictor of caries after age and was a more powerful predictor than the frequency with which children's teeth were reported to be brushed. A major conclusion was that the adverse impact of certain sugar-containing foods appeared to be restricted to children who brushed their teeth once a day or less. This agrees with earlier work by Schroder and Granath (1983) that clean teeth, irrespective of dietary habits, could be regarded as 'no-caries risks'. The authors concluded that the findings implied that for pre-school children, advice to brush twice per day with a fluoride toothpaste may be more effective in preventing caries than advice to restrict sugars.

Some studies carried out on older cohorts with similar methodology have included: Dominguez-Rojas et al, 1993; Mattiasson-Robertson and Twetman, 1993 and Dummer et al, 1990.

In this section work involving a multi-disciplinary approach to risk assessment has been described. The UNCCRAS was a unique, comprehensive longitudinal study which resulted in a set of prediction models for caries risk. It was, however, carried out on schoolchildren and this thesis has focused on risk assessment of pre-school children. The work in Sweden by Grindefjord and co-workers involved a population with a high proportion of immigrants which has little direct relevance to Scotland as a whole. They found that one of the highest risk factors was immigrant status, a factor which may mask other important predictors, such as frequency of sugar intake. A major problem is, therefore, geographical generalisability of risk models. A second problem has concerned the statistical analysis and methodological techniques of data collection. These do not tend to be standardised to allow easier comparison and interpretation of the results. Hausen (1997) pointed out the

pertinent pieces of information that should be available in a good caries prediction study report including: baseline caries score; sensitivity; specificity; positive predictive value; negative predictive value; and a logistic regression model. These methods should be understood in order to evaluate the report critically. Unfortunately, very few of studies reported have adhered to this. Methodological differences include: microbiological techniques (see section 2.2.4 and chapter 4); collection of socio-demographic, dietary and dental health behaviour data (see section 2.2.5, 2.2.6, 2.2.7 and chapter 4) and caries diagnosis methods (see section 2.2.3 and chapter 4). Many studies have used different and several methods of statistical analysis (see section 2.2.2). The methodological techniques adopted in the study for this thesis will be described in chapter 4. Statistical analysis involved both novel and traditional methods of risk model development to allow comparison with other longitudinal studies.

2.2.10.3 Summary of multiple factors

The literature review on multiple factors aimed to assess the conclusions of authors using multiple factors and the relative importance of these factors in caries prediction. Krasse (1988), in a review, concluded that the best prediction of caries activity is obtained by combinations of factors which are important contributors to the pathogenesis of dental caries (a so-called aetiological risk model). However, many authors have highlighted the importance of non-aetiological factors such as socio-demographic characteristics (Hunt, 1990, Davenport, 1990). Demers et al (1990), in a review, noted that studies considering only one factor at a time did not

take into account the multifactorial aetiology of dental caries and previous studies indicated that a single test is not sufficient to predict a high proportion of caries risk children. However, many authors although appreciating the importance of a multifactorial approach, have expressed concern regarding the lack of success in predicting caries (Fejerskov, 1990; Eriksen and Bjertness, 1991; Hausen et al, 1994 and Hausen, 1997). This concern is supported by the literature review provided in section 2.2.10.

2.3 Outline and aims of thesis

This thesis describes a 4-year longitudinal prospective study carried out in order to assess the feasibility of a partnership with health visitors to identify high caries risk pre-school children (4-year-olds). A multi-disciplinary approach involving dental, microbiological, and socio-demographic factors was employed. There were two main strands of the study. The first related to the feasibility of a partnership with health visitors and the second involved the development a 'high risk' caries prediction model for 4-year-old pre-school children.

Although a complete review of the literature was deemed essential in terms of both individual and multiple factors in caries risk assessment, this thesis will focus on the development of a caries risk prediction model for 4-year old pre-school children using data collected on various factors. These factors and the methodology of data collection will be described in chapter 4. The factors investigated were namely, dental, microbiological, health behaviour, socio-demographic (including medical) and hunch factors.

Therefore, the aims of this thesis were:

1. To assess the feasibility of a partnership with health visitors (existing health services personnel) to access pre-school children in order to collect dental, microbiological, health behaviour and socio-demographic data at ages 1, 2, 3 and 4-years (chapter 3) and

2. To develop a novel model for risk assessment of 4-year old pre-school children which could be used in a community setting to allow targeted preventive care (prior to irreversible tooth destruction) to those children at high risk of developing dental decay (chapters 4, 5 and 6).

Chapter 3: Feasibility of employing existing health visitors for the purpose of caries risk assessment of pre-school children

3.1 Introduction

This chapter was designed to introduce the reader to the multidisciplinary nature of health visiting and the health visitor's role within the community. This part of the study for the thesis focused on the ability of health visitors to access pre-school children within their daily timetable for the purpose of gaining consent for a longitudinal caries risk assessment study and the collection of risk assessment data (in partnership with a study dentist). The collection of caries risk assessment data for pre-school children by health visitors, to allow identification of those minority of children with the majority of the decay in Scotland (Chapter 1.1.3), could, in turn facilitate future targeting of preventive care at these caries risk children. This 4-year, prospective, longitudinal study was based in the city of Dundee, situated on the east coast of Scotland, directly north of the river Tay estuary. Dundee has a population of circa 170,000 people (population census, 1995), 22 main medical practices to which the 57 health visitors were attached and an annual average 2000 live births in the main hospital, Ninewells Hospital and Medical School. For the purposes of this study, the calendar year 1 April 1993 to 31 March 1994 was selected for investigation, a year during which a total of 1981 live births were recorded.

3.1.1 Introduction to the health visitor (HV)

The profession of health visiting was first established in 1862 (Simmonds, 1965). At this time and for many years, health visiting involved meeting basic family needs and prevention of the many diseases prevalent at that time, for example, dysentery. Nowadays, health visitors are registered general nurses with a post registration qualification in health visiting, which has recently been converted to a degree course. Many have a variety of other qualifications, the most common being midwifery. They are registered nationally with the United Kingdom Central Council for nurses, midwives and health visitors (UKCC) and are usually employed by the National Health Service. In Dundee, the health visitors are attached to the general medical practices and have a case-load drawn from the medical practitioners' lists. While this ought to mean the list comprises the entire population registered with that general practitioner, in reality it means the families with children under five years of age and some elderly people. Health visitors in this system are then able to undertake a detailed caseload analysis from which to identify a range of priorities (Luke and Orr, 1992).

3.1.2 Sphere of activity of the health visitor

Health visitors are nurses whose work is fundamentally about prevention of illness rather than cure. They are based in the community rather than hospital and carry out very few nursing procedures. Their remit, however, is broad and with such widespread access to the families and individuals which make up any community they have considerable potential to influence health within this stratum of society.

Wider community involvement also means being involved in community development, which is seen as bringing about change by consensus, and may even mean community action, which seeks to bring change by conflict. It means urging demands on local or central government and on professionals for innovations or change in the pattern of health provision and the allocation of resources, for example, in local playgroup provision (Luke and Orr, 1992). The main tasks of the health visitors in Dundee can be broadly divided into: health education and promotion; antenatal visiting and advice; child growth and development monitoring; elderly, bereavement and handicapped visiting; accident prevention; as well as immunisation and special clinics, including sleep, obesity, menopause and asthma (Tayside Health Board leaflet, Appendix 3.1). Health visiting is not task orientated, however, and should be innovative and pro-active. Health visitors treat people holistically and as members of the families and communities in which they live. They are independent 'practitioners' responsible for their own case-load and prioritise, organise and carry out their own work. As the title health visiting suggests, much of their time involves visiting homes within the community – indeed, they are the only health professional visiting well people in their own homes.

3.1.3 Work location of the health visitor

The work location of the health visitor is varied and dependent upon the task involved. Their base is the general medical practice and within these practices or health centres are held various clinics, including baby clinics, child developmental screening clinics and immunisation clinics. Much of the health visitor's daily direct

contact with the community, however, is in the homes of the individual families concerned. These home visits are made to ante-natal mothers, new mothers, pre-school children (and their families), and the elderly. Health visitors also visit individuals in hospitals, schools, nurseries, community centres and the homes of childminders.

3.1.4 Aims

For the purposes of this study the focus was placed on the health visitors' role with children, particularly that pertaining to access of pre-school children and child growth and development monitoring. The fact that every ante-natal mother (and, therefore, every child) in the United Kingdom is allocated to a health visitor and each child is thereafter monitored developmentally by that health visitor, was a critical factor in the decision to attempt to employ this means of access to pre-school children.

The main aims of the part of the study covered in this chapter were threefold:

1. To determine the extent to which health visitors could be recruited to participate in a longitudinal caries risk assessment study of pre-school children.
2. To determine the extent to which health visitors in Dundee could gain consent for a 4-year longitudinal caries risk assessment study of pre-school children in Dundee.
3. To determine the extent to which health visitors in Dundee could gain access to a consented cohort of pre-school children at ages 1-, 2-, 3- and 4-years for

the purpose of collection of caries risk assessment data in partnership with a study dentist.

3.2 Health visitors and dental caries in pre-school children

As previously noted, health visitors maintain a unique position in the community through contact with mothers and their children from before birth until school age. It has been recognised that much of the dental disease in children occurs long before they come into contact with the dental profession (Todd et al, 1985, Hinds & Gregory, 1995). Health visitors, therefore, have a key role to play in the early diagnosis and prevention of that disease. Health visitors, however, are busy people with changing priorities on a daily basis. It may, therefore, be difficult to fit oral health in with other competing pressures.

3.2.1 Relationship to the Nuffield Report

The Nuffield Foundation's publication entitled 'Education and Training of Personnel Auxiliary to Dentistry' (Tyrell, 1993) emphasised the ever increasing role to be played by auxiliaries in delivering dental and oral health care. It revealed a need for reappraisal of the idea of 'dental teams' and adoption of a multidisciplinary approach to the development of oral health professional and support personnel appropriate to national needs in both quality and quantity. Health visitors are key members of this multi-disciplinary team aimed at achieving a reduction in dental decay both in Scotland and in the United Kingdom as a whole. Indeed, the Bloomfield report in 1992 stated that, "there seems to be an overwhelming national

interest in setting all the children of the country on a sound dental path". In the view of the Nuffield Foundation, the most effective way of doing this would be to use as a basis the machinery already in existence for health checks of young infants provided by health visitors. The Nuffield report states that the introduction of simple and easily carried out diagnostic tests as part of a screening programme might be the best way of identifying children at risk. A main aim of this study and thesis was to investigate the feasibility of a partnership with health visitors to carry out caries risk assessment using such simple tests, including microbiological saliva sampling and questionnaire data (see chapter 4). The results from these tests will be discussed in chapter 6. Although health visitors are ideally placed to provide a screening and / or risk assessment for caries in pre-school children, it is mandatory that those identified as high risk are followed through within the dental care system. To allow this, communication must exist between dentist and health visitor and a multidisciplinary approach should be the aim (Bentley and Holloway, 1993; Quinn and Freeman, 1994). A more recent move toward a multidisciplinary approach to caries prevention in the community was implemented in Scotland in 1996 (Stephen & Hesketh, 1996). This comprised a caries-prevention pack designed for use by general dental practitioners, health visitors, general medical practitioners and community pharmacists. The three main components of core information, practical tips and resources for use in daily practice were aimed at providing a clear and consistent message to both the health care professionals and the public regarding caries and its prevention. The long term goal was to help to achieve the target set by

the Scottish Office for decay levels for 5 year olds to be reached by the year 2000 (Oral Health Strategy for Scotland, 1995).

3.3 Hypotheses of chapter to be tested

Hypothesis 3.1

Health visitors in Dundee can be **recruited** to participate in a 4-year longitudinal caries risk assessment of pre-school children.

Hypothesis 3.2

Health visitors can gain **consent** for a 4-year longitudinal caries risk assessment study of pre-school children.

Hypothesis 3.3

It is **feasible** to employ existing health visitors to collect caries risk assessment data (involving microbiological saliva sampling and questionnaire completion) for the majority of a large cohort of pre-school children for a 4-year longitudinal caries risk assessment study.

3.4 Materials and methods

3.4.1 Study design and initial set up

3.4.1.1 Introduction

The first proposals for the study were collated in February 1992. These were fully outlined in an application for funding to the Chief Scientist Office, Scottish Home

and Health Department. Subsequent to minor modifications, funding was obtained in October 1993 and the study dentist recruited in November 1993 to take up employment on 1 January 1994. The target cohort consisted of all those children born and resident in Dundee between 1 April 1993 and 31 March 1994. Data collection, therefore, began on April 1 1994 when the first consented study children were aged 1-year. This allowed for a three-month set up period at the beginning of the study and a three-month wind down period at the end to ensure completion of data collection.

3.4.1.2 Recruitment of health visitors

In order to recruit the health visitors to participate in the study the director of nursing services for Dundee was approached in 1992. A subsequent meeting was held between the study team and the three clinical nurse managers responsible for the day to day running of nursing services in Dundee. At this time a health visitor was appointed as a liaison between the study team and the health visitors. The liaison health visitor was included in the formulation of grant proposals and a grant holder on the study. The meeting with the clinical nurse managers resulted, in 1993, in a series of consultation meetings with all the 57 health visitors working in Dundee. These consultations enabled the study team to state the aims of the study and discuss how these could best be met. The level of commitment required to carry out the tasks for the study was emphasised, as these would be additional to the daily duties of each health visitor. The health visitors were invited to comment on any aspect of the study design and discuss issues pertaining to the study.

3.4.1.3 Consent for study children

Written consent for each child to participate in the study was obtained from the parent or guardian by the health visitor at the time of the 8-month developmental screening. The consent form was presented to the parent or guardian of the child in the form of a brief written explanation of the study, followed by a reply slip on which they could consent or decline to participate (Appendix 3.2). Three copies of the signed consent form were obtained. The parent or guardian of the child and the child's health visitor each retained a copy. The third copy was returned to the study team. As consent was obtained at the time of the child's 8-month developmental screening, the first consent forms were required for use in December 1993 for those children born in April 1993. The list of children for the study cohort was obtained from the Child Health Department of Strathmartine Hospital in Dundee. These lists contained details of the child's name, address, date of birth, general medical practitioner, health visitor and CHI number on a month to month basis. The child's Child Health Index (CHI)) number, which included the date of birth and a further four digits, was unique to each child. This was, therefore, used as the study number to identify each individual study child. This ten-digit number was used for all correspondence pertaining to that child for the 4-year duration of the study. The consent form showed the child's name, address and study number. These were sent directly to each appropriate health visitor one month prior to the date of the child's 8-month developmental screening with a covering letter and list containing details of all possible children eligible for recruitment.

3.4.1.4 Set up of the laboratory

A microbiological laboratory was mandatory to provide support for the oral microbiological saliva sampling aspects of the caries risk assessment carried out by the health visitors. A room was chosen in the laboratory area of the Dental School in Dundee and refurbished into a basic laboratory. Purchase of equipment necessary for microbiological saliva sampling, microbiological processing and microorganism identification was carried out before April 1994 when saliva sampling of the children at 1-year of age was due to begin. Such equipment included an -80^{0}C freezer, rotamixer, incubator and pH meter and colony counter (Appendix 3.3). A full inventory is outwith the scope of this chapter and the reader is referred to chapter 4.2 for microbiological methodology.

3.4.1.5 Training of health visitors

In February 1994, four training sessions were held for the health visitors by the study team. These were organised to discuss the most effective ways of accessing the children and obtain feedback on draft questionnaires. The technique of saliva sampling was demonstrated by the study dentist, practised by the health visitors and the logistics of sampling kit provision and collection discussed (Appendix 3.4). The preliminary results of consent rates for the first two months of children born were discussed and the success of these provided a source of encouragement to the health visitors and the study team at this time.

3.4.2 Pilot study

A pilot study, which involved 7 volunteer health visitors and 7 children, was carried out prior to implementation of the study. One child was chosen by each of the health visitors from their own available case-load and, subsequent to consent being obtained from the parent or guardian, a sample of saliva, parental questionnaire and health visitor questionnaire was completed for each child. The saliva samples were processed and microorganisms cultured and identified.

3.4.3 Dental examination

The complete methodology of the dental examination will be described in Chapter 4.1. Dental examination was carried out by the study dentist as close as possible to the child's birthday in each of the four years.

3.4.4 Access methods

The various methods used to access the children were grouped into five main categories, according to how the child was seen for the dental examination by the study dentist. These access methods revealed whether or not the health visitor and study dentist accessed the children jointly or separately. Firstly, a joint home visit (JHV) entailed the health visitor and study dentist visiting the child's home together, where saliva sampling and questionnaire completion were carried out consecutively. A separate home visit (SHV) involved the study dentist making a separate arrangement to visit the child's home for dental examination, the saliva sample having been taken at a previous time by the health visitor. This visit was arranged

either by letter or telephone and no opportunistic visiting was carried out. If saliva sampling and dental examination were carried out together at a clinic or health centre, this was termed a joint clinic visit (JCV), but if dental examination was completed independently by the study dentist at a health centre or clinic, this was termed a separate clinic visit (SCV). The fifth and final category was termed other visit (OTH) and included joint or separate visits by the study dentist and health visitor to other locations such as nurseries, childminders, community centres and hospitals.

3.4.5 Saliva sampling

A complete description of the methodology of the microbiological aspects of the study is detailed in Chapter 4.2. The technique of saliva sampling used was the tongue-loop method described by Beighton in 1986. This was a quick and simple method of obtaining a loop of saliva from the tongue of a young child and one readily adopted by the health visitors (Appendix 3.5).

The study dentist carried out saliva sampling of the mothers' of the children in the first year of the study.

3.4.6 Questionnaires

The study questionnaires were used to collect data regarding the children in each of the 4-years of the study. These were a parental questionnaire (PQ) (Appendix 3.6) and a health visitor questionnaire (HQ) (Appendix 3.7) and will be fully described in chapter 4.3.

3.4.7 Health visitor feedback questionnaire (HVFQ)

This questionnaire, distributed annually in the latter three years of the study to the health visitors, aimed at assessing their views on the study design and progress (Appendix 3.8). Constructive criticism was encouraged and used to facilitate any minor modifications of study design required. A question on the health visitor's opinion of dental health as a priority was also included each year. This questionnaire was anonymous to allow the health visitors to express their opinions freely.

3.4.8 Initiatives to maintain health visitor motivation

Prior to implementation of the study, an information leaflet was provided for each health visitor (Appendix 3.9) which contained a detailed explanation of the procedures involved and a contact number for any queries. A laminated flow diagram was also distributed (Appendix 3.10) which illustrated at a glance the procedure of oral microbiological saliva sampling. This was sized to fit into the health visitors' diary. In addition, bimonthly newsletters were sent to each health visitor (Appendix 3.11). These provided updates on the study progress, any pertinent issues and forthcoming events. An annual buffet lunch was also held for all health visitors and management staff. These were informal sessions that allowed open communication between all members of the study team and the health visitors and also the opportunity to give a brief and personal update on the study progress. Finally, Christmas cards were sent annually to all the health visitors (Appendix 3.12).

The study dentist attended many of the monthly meetings held for health visitors by health board management. This allowed current issues or queries to be discussed. Regular contact was made with the health visitors to update them on their progress in terms of the study children. Individualised lists were collated on the databases that provided each health visitor with a list of data outstanding. A continued effort was made to maintain both health visitor motivation and maximum data collection. Newly employed health visitors were trained by the study dentist on a needs basis and the study dentist also gave tutorials to trainee nurses and health visitors and provided a lecture at the local college for student health visitors.

3.4.9 Modifications and developments during the study period

3.4.9.1 Microbiological saliva sampling and processing

Following a period in the early months of the study when there was contamination of media plates, a laminar flow cabinet was purchased for the laboratory and no further contamination problems were experienced.

Initially, four health centres were chosen as pick-up and drop-off points for the saliva sampling kits. Due to demand by the health visitors, a supplemental health centre was provided in the north east of the city. Following a review period, this was retained.

3.4.9.2 Questionnaires

The questionnaires were modified slightly from the first year of the study to allow ease of data entry. A box system was introduced and the database set-up accordingly. This did not alter the questions, merely the style in which they were answered. The colour of the parental questionnaire was changed each year from yellow in the first year to green in the second, pink for the third year and finally blue for the fourth year. Many questions in the first health visitor questionnaire, for example ethnic origin, were only asked in the first year of the study, as it was not time-dependent data which would alter throughout the study duration. Subsequent questionnaires were, therefore, shorter.

3.5 Results

3.5.1 Recruitment of health visitors

All the fifty- seven health visitors for the city of Dundee agreed to participate in the study from its outset, which gave a recruitment rate of 100 percent.

3.5.2 Training of health visitors

Four training sessions in total were held for the health visitors prior to the start of the study. A total of 50 out of 57 health visitors attended at least one training session, an attendance rate of 88%. For those seven who did not attend, some were contacted directly by the study dentist for training, whilst the remainder agreed to be trained by a health visitor who had attended a training session.

Results and actions points from the training sessions can be summarised as follows:

1. Final adjustments were made to parental and health visitor questionnaires.
2. Un-named consent forms were sent to health visitors to allow opportunistic consent to be obtained at times other than the 8-month developmental screening.
3. A fourth additional location for igloos (insulated plastic containers) containing fresh and used sampling kits was provided in the west-end of the city.
4. An information leaflet for the study was distributed to all health visitors.
5. Access to the children would be gained at clinics / health centres, if possible, to allow dental examination by the study dentist at the same time as saliva sampling and questionnaire completion by the health visitor. If this was not feasible, other means of access would be used.

3.5.3 Consent rate

3.5.3.1 Number and percentage of children consented

The consent rate varied over the 4-year period. The figures for the first & second years and third & fourth years of the study are shown in Table 3.1. It should be noted that the figures for years 1 and 3 were identical to years 2 and 4 respectively due to the method of data entry into the database. The consent rate was calculated as the number of children for whom written consent had been obtained as a percentage of available cohort (all those children born and resident in Dundee, excluding those without consent who had moved outwith the city and deceased). Those children

who had refused, withdrawn, and for whom no reply had ever been obtained were not included in the consented total. The total consent rate for years 1 and 2 of the study was 89% and for years 3 and 4, again, 89%.

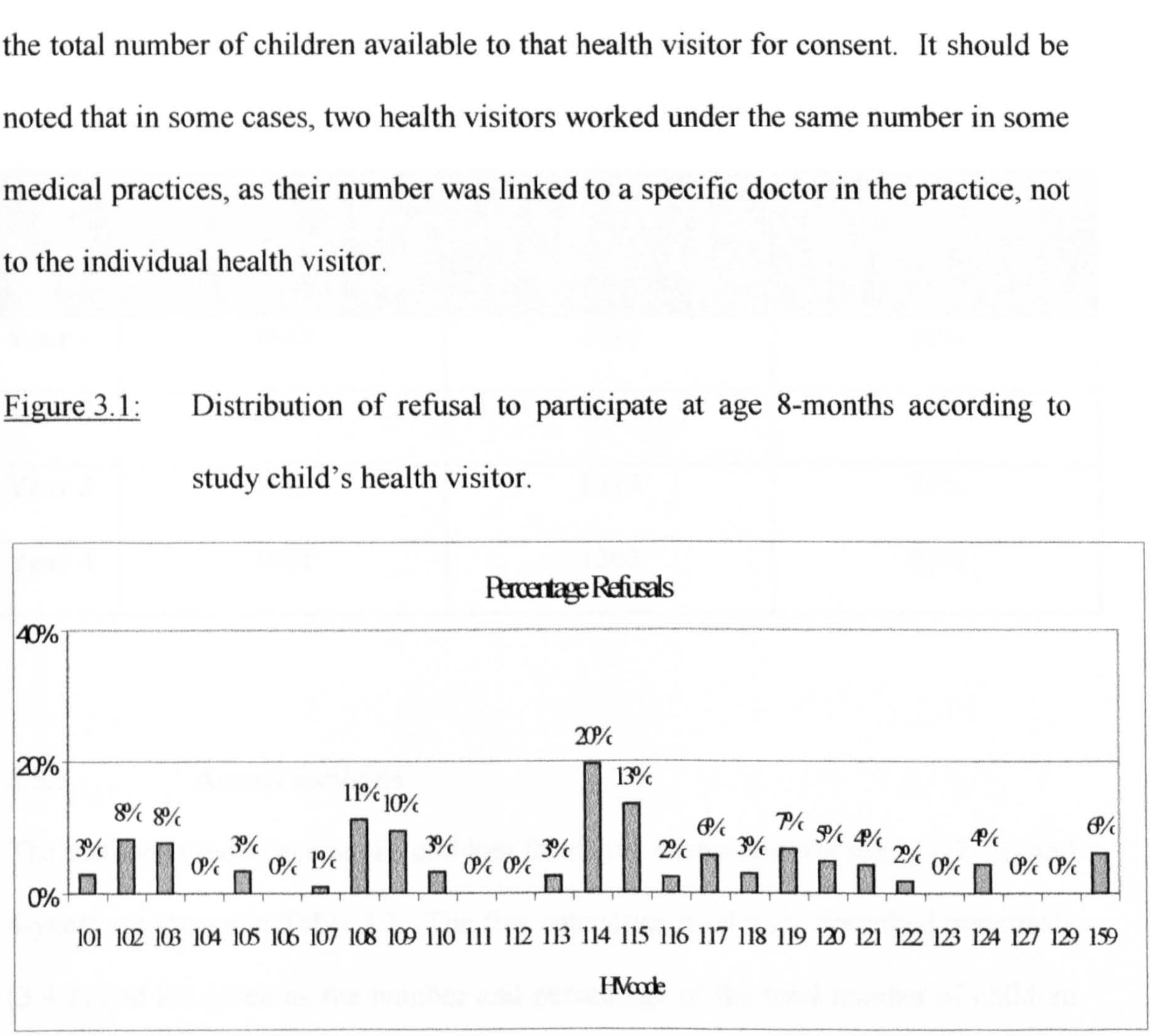
Table 3.1: Number and percentage of consented children for the four-year study duration.

	Number of children	
	Years 1 and 2	**Years 3 and 4**
Born in Dundee (01.04.93 - 31.03.94)	1981	1981
Consented	1532	1500
Refused	93	92
No Reply	95	93
Withdrawn	19	22
Moved and consented	151	181
Moved not consented	89	91
Deceased	2	2
Total consented	**1683**	**1681**
Available cohort	**1890**	**1888**
Consent rate	**89%**	**89%**

3.5.3.2 Distribution of refusal to participate according to the child's health visitor

Figure 3.1 shows the percentage distribution of refusal of parent / guardian consent according to the health visitor present at the 8-month developmental screening. This reflects the number of refusals received from each health visitor as a percentage of the total number of children available to that health visitor for consent. It should be noted that in some cases, two health visitors worked under the same number in some medical practices, as their number was linked to a specific doctor in the practice, not to the individual health visitor.

Figure 3.1: Distribution of refusal to participate at age 8-months according to study child's health visitor.



3.5.4 Dental examination

The number and percentage of children for whom a dental examination was carried out is shown in Table 3.2.

Table 3.2: Number and percentage of children for whom a dental examination was carried out for each of the four years of the study.

	Number of consented Children	Number of dental examinations	Percentage examined
Year 1	1683	1419	84%
Year 2	1683	1394	83%
Year 3	1681	1219	73%
Year 4	1681	1365	81%

3.5.5 Access methods

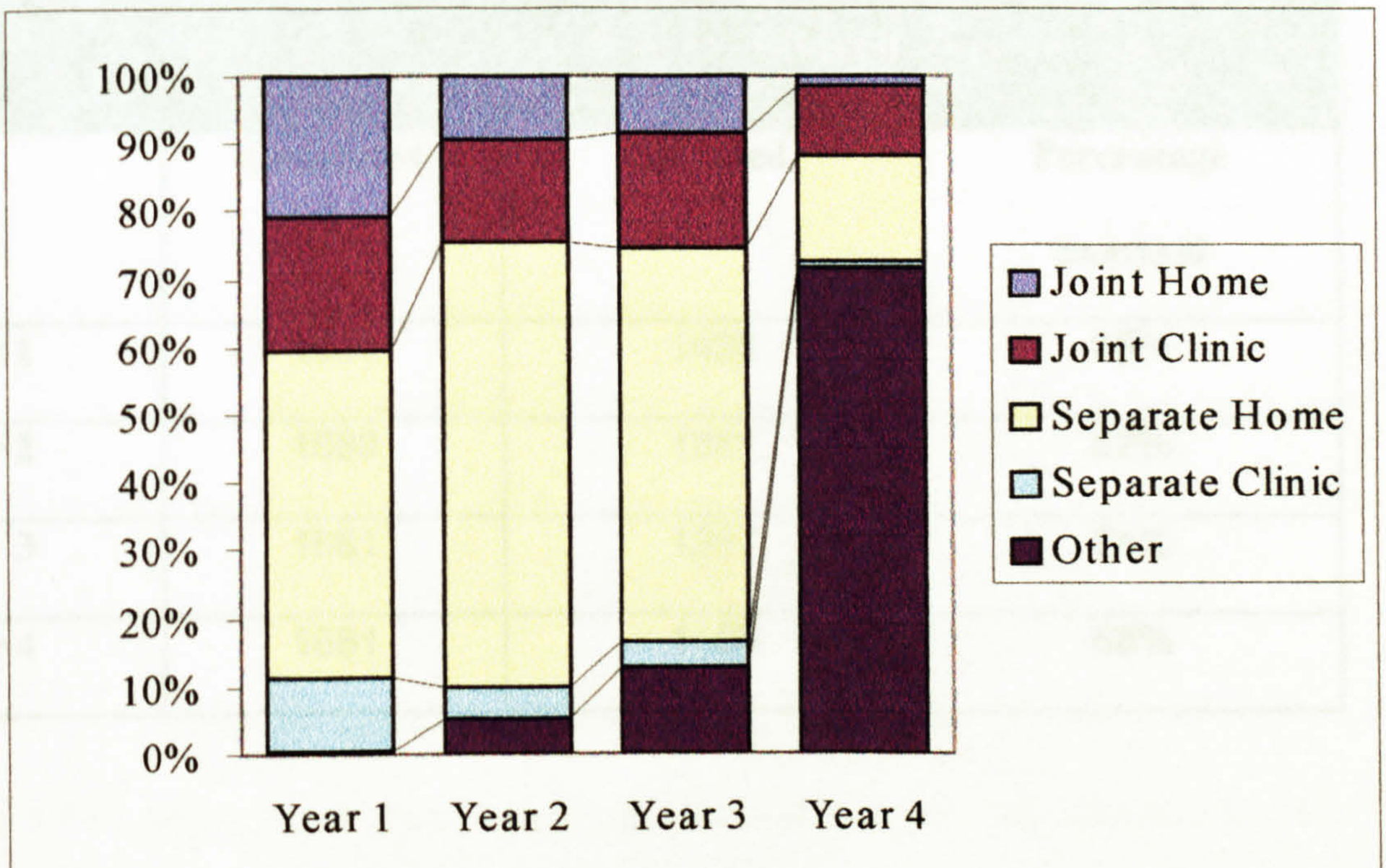
The methods used to access the children for dental examination at ages 1-, 2-, 3- and 4-years are shown in Table 3.3. The five categories used were described previously (3.4.4) and are given as the number and percentage of the total number of children dentally examined. Figure 3.2 shows the percentage distribution of the access methods in the four years of the study. The changes in the types of access methods used for the study duration were highly statistically significant using a 4x5 chi-squared test to the $p<0.001$ level (Pearson Chi-Square = 2899.8). The 'other' visits

changed significantly (Pearson Chi-Square = 2605.8) and this change was also significant linearly (Linear-by-Linear Association = 1977.4). Joint and separate visits changed significantly with respect to each other (Pearson Chi-Square = 87.97) but most of this change was non-linear (Linear-by-Linear Association = 11.12). Home and clinic visits changed significantly (Pearson Chi-Square = 70.8) and again this change was not linear (Linear-by-Linear Association = 0.16).

Table 3.3: Methods used to achieve access to the children for dental examination in each of the four years of the study.

	Number of children			
	Year 1	**Year 2**	**Year 3**	**Year 4**
Dentally examined	1419	1394	1219	1365
Joint home visit	292 (21%)	130 (9%)	104 (8%)	21 (2%)
Joint clinic visit	285 (20%)	212 (15%)	204 (17%)	138 (10%)
Separate home visit	680 (48%)	912 (66%)	711 (58%)	216 (16%)
Separate clinic visit	158 (11%)	65 (5%)	43 (4%)	10 (<1%)
Other	4 (<0.1%)	75 (5%)	157 (13%)	983 (72%)

Figure 3.2: Distribution of access methods used in each of the four years of the study as a percentage of the total number of children accessed for dental examination.



3.5.6 Microbiological saliva sampling

3.5.6.1 Child microbiological saliva samples

Table 3.4 shows the number and percentage of children at ages 1, 2, 3 and 4 years for whom microbiological saliva samples were obtained.

Table 3.4: Number and percentage of children for whom microbiological saliva samples were obtained for each of the four years of the study.

Number of children			
	Consented	**Sampled**	**Percentage Sampled**
Year 1	1683	1436	85%
Year 2	1683	1381	82%
Year 3	1681	1247	74%
Year 4	1681	1150	68%

3.5.6.2 Parental microbiological saliva samples

1170 parental samples were obtained by the study dentist in the first year of the study.

3.5.7 Questionnaires

3.5.7.1 Health visitor questionnaire (HQ)

The results from the numbers of completed health visitor questionnaires are shown in Table 3.5. The number and percentage of these questionnaires completed by the health visitors for each of the 4 years of the study are given.

Table 3.5 Number and percentage of health visitor questionnaires (HQ) completed for each of the four years of the study.

	Number of children Consented	Number of HQ's Returned	Percentage HQ's Returned
Year 1	1683	1426	85%
Year 2	1683	1394	83%
Year 3	1681	1261	75%
Year 4	1681	1163	69%

3.5.7.2 Parental questionnaire (PQ)

Table 3.6 shows the number and percentage return of parental questionnaires for the 4- year duration of the study. These are shown as a percentage of the number of children consented for each year of the study.

Table 3.6 Number and percentage of parental questionnaires (PQ) completed for each of the four years of the study.

	Number of Children Consented	Number of PQ's Returned	Percentage of PQ's Returned
Year 1	1683	1405	83%
Year 2	1683	1342	80%
Year 3	1681	1250	74%
Year 4	1681	1149	68%

3.5.7.3 Health visitor response to children caries risk status

The response rate to the question contained in the health visitor questionnaire 'is the child at high risk of developing dental caries yes/no? is shown in Table 3.7

Table 3.7: Health visitor response rate to question on childrens' caries risk status.

	Number of HQ's returned	Number of questions answered	Percentage response
Year 1	1426	1026	72%
Year 2	1394	1134	81%
Year 3	1261	1169	93%
Year 4	1163	988	85%

3.5.8 Distribution of saliva sample and questionnaire returns by health visitors

The following set of results have been provided to show the distribution of data collection by the health visitors in the study.

3.5.8.1 Returns in year-1 of study

Figure 3.3 shows the percentage return of child saliva samples (CS), health visitor questionnaires (HQ) and parental questionnaires (PQ) for the first year of the study.

3.5.8.2 Returns in year-2 of study

Figure 3.4 shows the percentage return of child saliva samples (CS), health visitor questionnaires (HQ) and parental questionnaires (PQ) for the second year of the study.

3.5.8.3 Returns in year-3 of study

Figure 3.5 shows the percentage return of child saliva samples (CS), health visitor questionnaires (HQ) and parental questionnaires (PQ) for the third year of the study.

3.5.8.4 Returns in year-4 of study

Figure 3.6 shows the percentage return of child saliva samples (CS), health visitor questionnaires (HQ) and parental questionnaires (PQ) for the fourth year of the study.

Figure 3.3: Percentage return of child saliva samples, health visitor questionnaires and parental questionnaires for year-1 of the study.

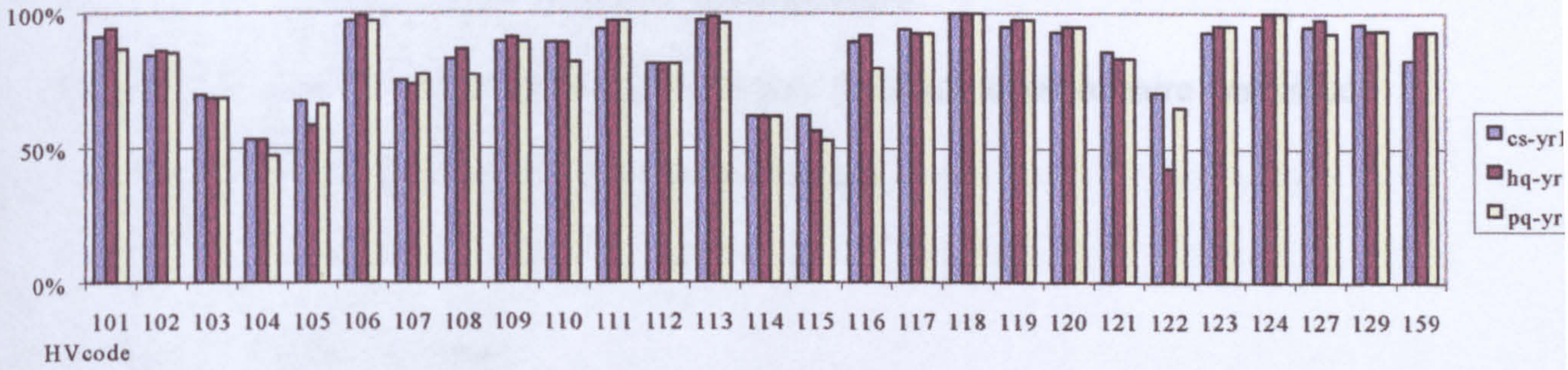


Figure 3.4: Percentage return of child saliva samples, health visitor questionnaires and parental questionnaires for year-2 of the study.

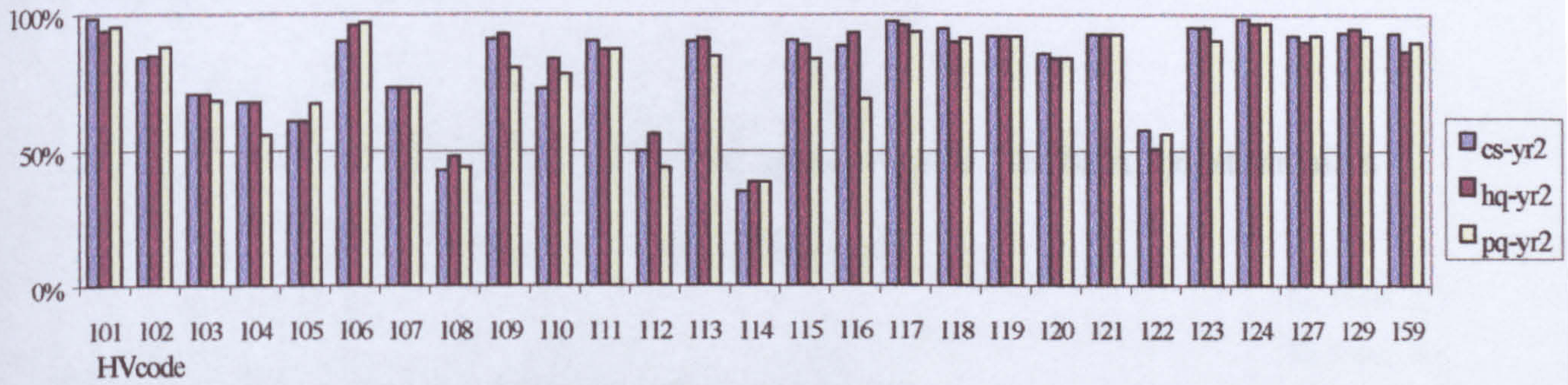


Figure 3.5: Percentage return of child saliva samples, health visitor questionnaires and parental questionnaires for year-3 of the study.

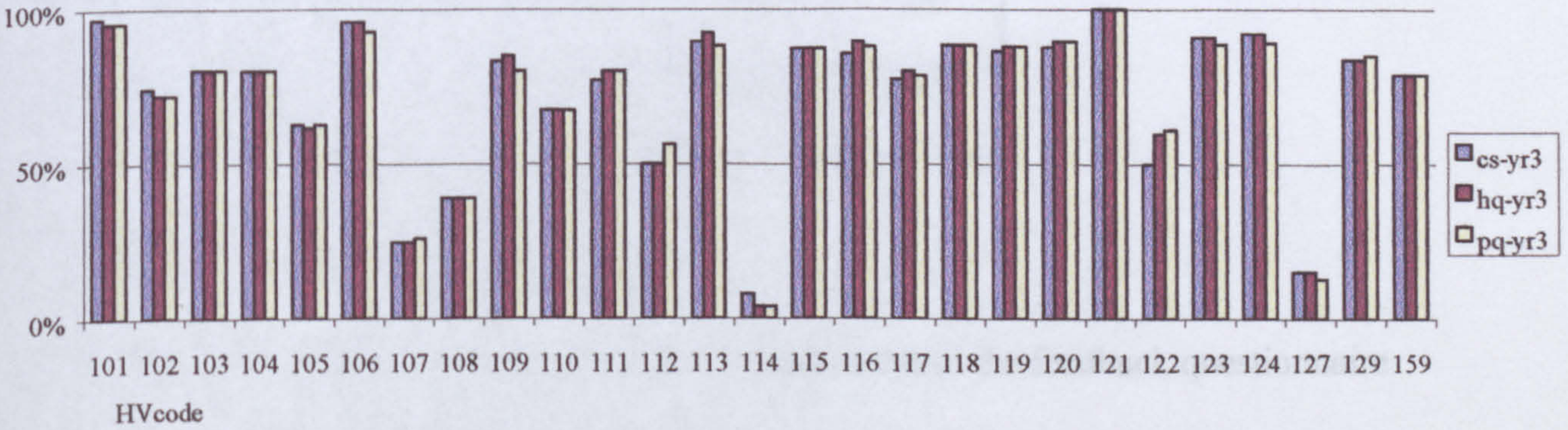
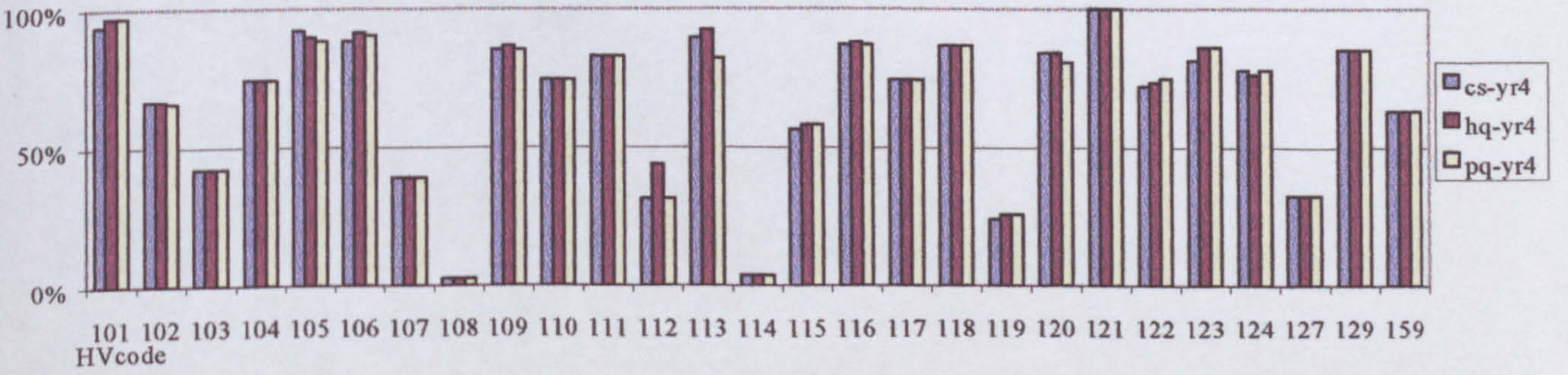


Figure 3.6: Percentage return of child saliva samples, health visitor questionnaires and parental questionnaires for year-4 of the study.



3.5.9 Health visitor feedback questionnaire

In years 2, 3 and 4 of the study a health visitor feedback questionnaire was issued anonymously. This section provides the main results.

3.5.9.1 Return rates

The results of the return rate of this annual feedback questionnaire are shown in Table 3.8.

Table 3.8: Number and percentage of health visitor feedback questionnaires returned in years 2, 3 and 4 of the study.

	Year 2	Year 3	Year 4
Number of HVs	57	62	59
Number of HVFQ's	43	45	38
Percentage return	75%	73%	64%

3.5.9.2 Results from questions

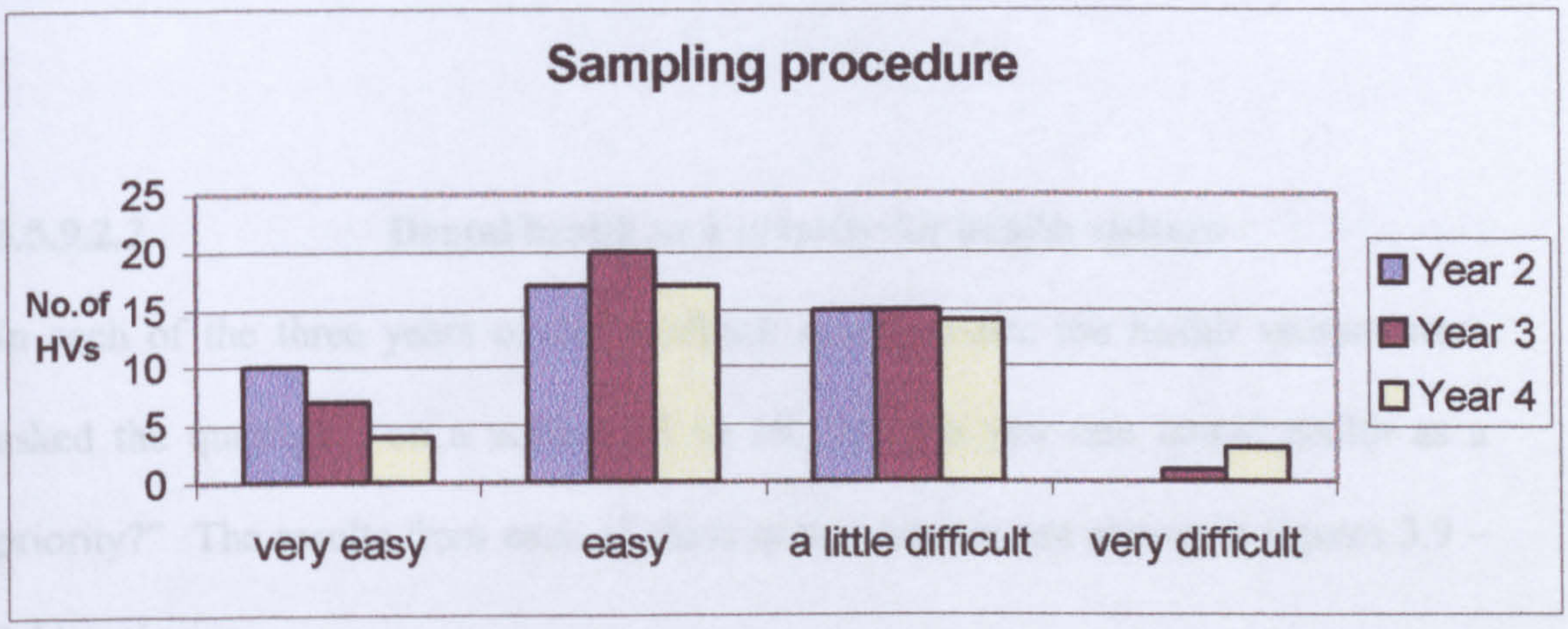
The results from the questions asked by the study team in the feedback questionnaire on the sampling procedure and health visitor questionnaire are shown in figures 3.7 and 3.8. These results reflect the level of satisfaction the health visitors had with these aspects of the study protocol.

3.5.9.2.1 Study methodology

3.5.9.2.1.1 Saliva sampling procedure

The results from the question asked in the feedback questionnaire, which related to the sampling procedure, are shown in Figure 3.7.

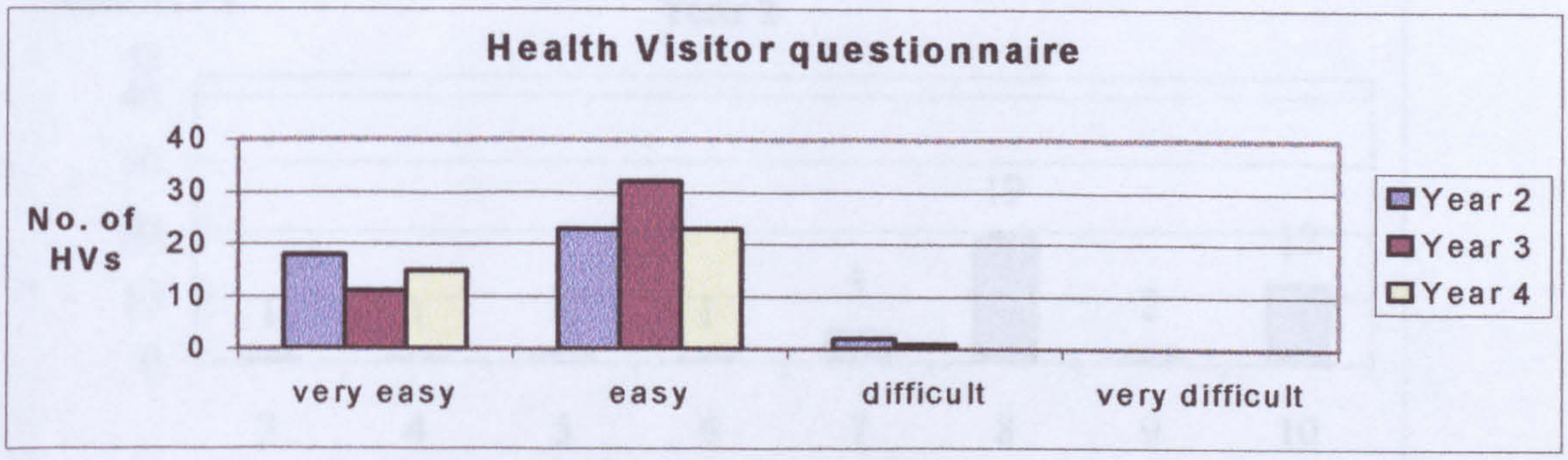
Figure 3.7: Results of the question entitled, "how did you find the sampling procedure?"



3.5.9.2.1.2 Study questionnaires

The results from the question asked in the feedback questionnaire, which related to the health visitor questionnaire, are shown in Figure 3.8.

Figure 3.8: Results of the question entitled, "how did you find the health visitor questionnaire?"



3.5.9.2.2 Dental health as a priority for health visitors

In each of the three years of the feedback questionnaire the health visitors were asked the question, "on a scale of 1 to 10, how do you rate dental health as a priority?" The results from each of these questionnaires are shown in figures 3.9 – 3.11 inclusive.

Figure 3.9: Results of question on dental health as a priority for health visitors in year-2 of study.

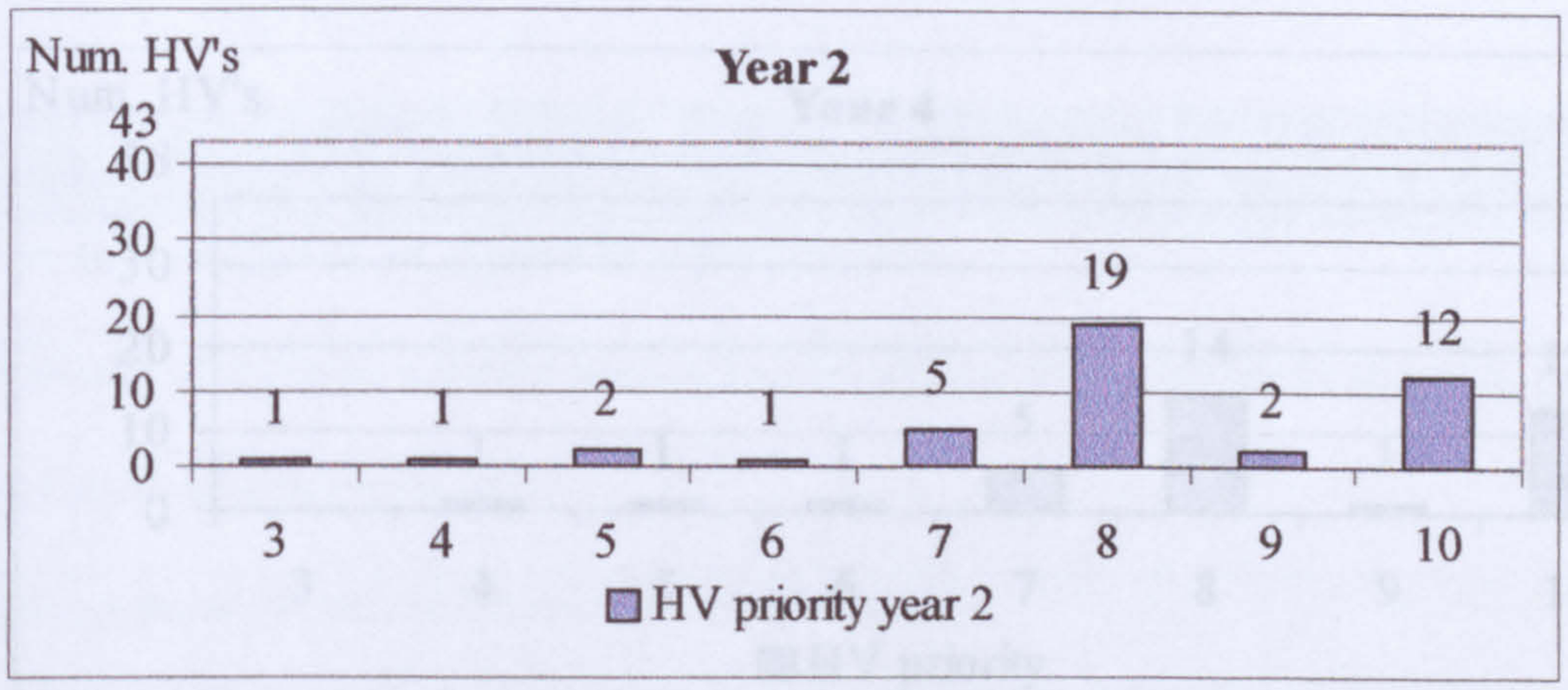


Figure 3.10: Results of question on dental health as a priority for health visitors in year-3 of study.

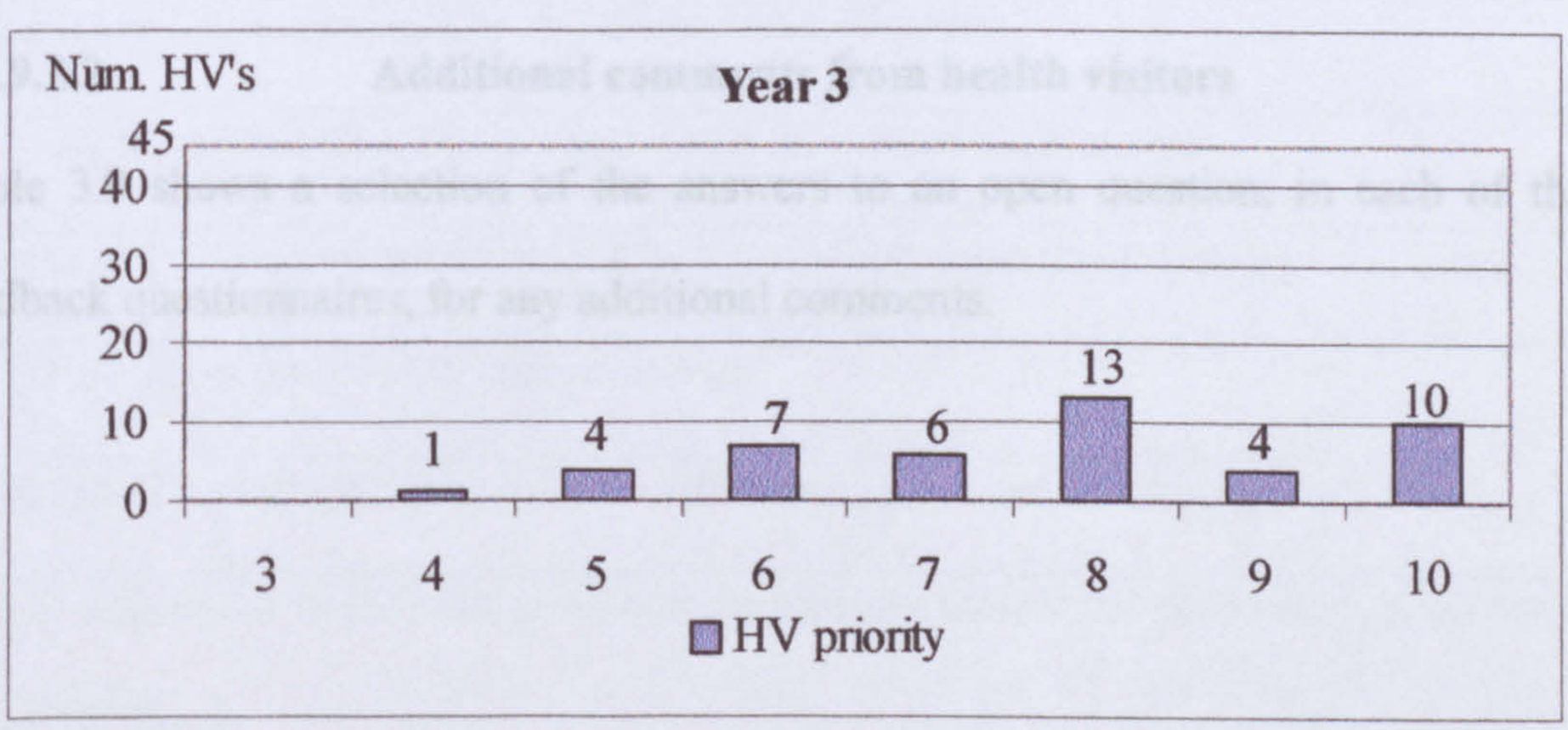
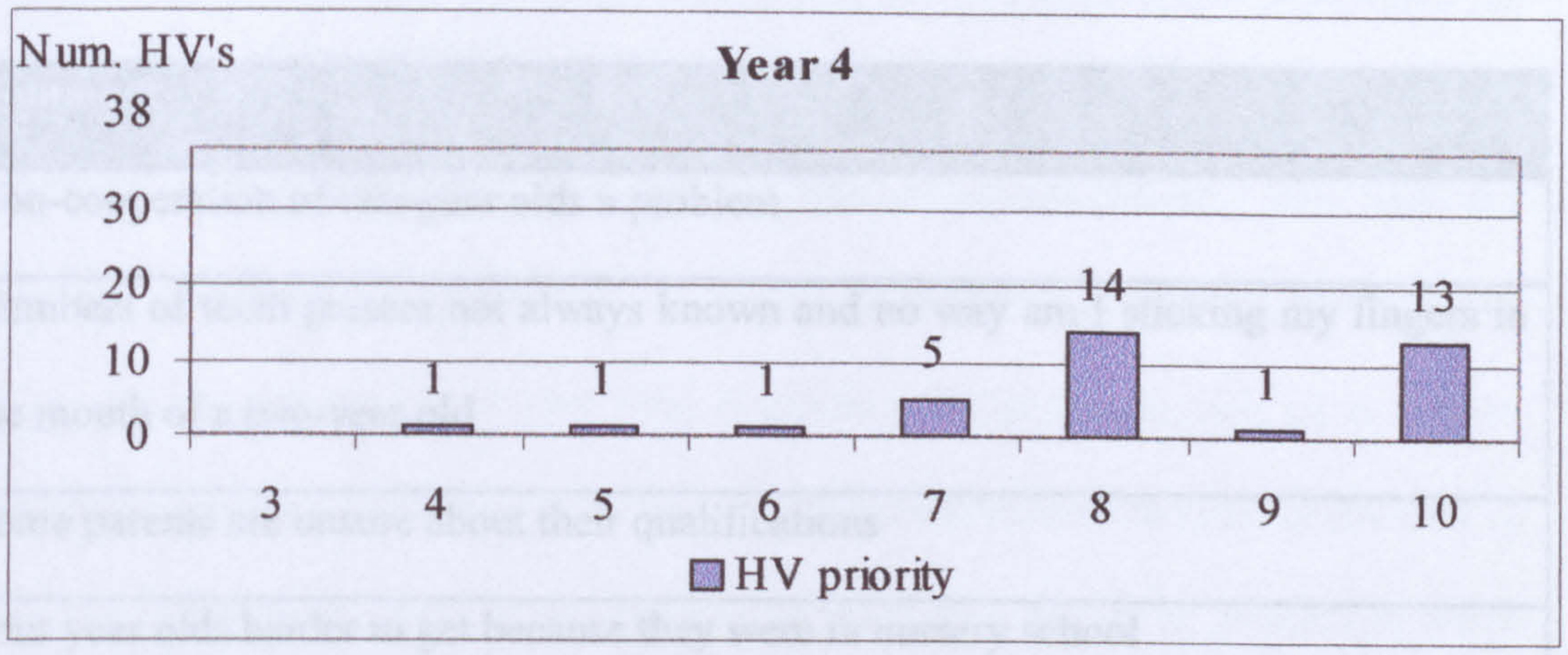


Figure 3.11 Results of question on dental health as a priority for health visitors in year-4 of study.



3.5.9.2.3 Additional comments from health visitors

Table 3.9 shows a selection of the answers to an open question, in each of the feedback questionnaires, for any additional comments.

Table 3.9: Results of the question for any additional comments from the health visitors on the any aspect of the study.

Comment
Non-cooperation of two-year olds a problem
Numbers of teeth present not always known and no way am I sticking my fingers in the mouth of a two-year old
Some parents are unsure about their qualifications
Four year olds harder to get because they were in nursery school
Parents did not like question about academic achievements
Is it necessary to bring in social class as it stigmatises unemployed and medicated?
Need more input ante-natally. Literature with pictures.
Difficult to contact children at age 3-years as mother back at work.
Parents not turning up for clinics or being in when appointment arranged at home.
Problems when new health visitor arrives.
Disappointing that all health visitor members of team not complying with study.
A well-organised study with good communication among all professionals involved. Good feedback.
The study is very worthwhile but it is having to take its place in a system of priorities, where other issues are more pressing.
Working mothers a problem.
I often wonder if the samples taken show bacteria, or if sample has been non-viable.

3.6 Discussion

3.6.1 Health visitors and dental health

The role of the health visitor in the community was outlined in the introduction to this chapter. As noted, they are uniquely placed in society in relation to the contact they have with ante-and post-natal mothers and pre-school children, thus have the potential to influence dental health. As noted by Williams and Fairpo (1984), they are in a unique position for they have a statutory obligation to visit all new-born children at home. The concept of health visitor involvement in oral health care is not a novel one, however. A document published by a group of workers in north west England entitled; 'Working together to promote dental health' outlined a campaign aimed at improving the dental health of young people. This was by focusing on young parents (and their children) and the health professionals in a position to contact and influence this target group (Bentley et al, 1992). Health visitors were recognised to be key health professionals in such a position. Many other authors have also regarded expectant mothers and those with very young children as a suitable target group for dental health education (Seward, 1967; Court Report, 1976; Blinkhorn, 1981 and Holt, 1985).

As early as 1965, Simmonds recommended a role of the health visitor in dental health education and this role has consistently been recognised since this time (Blinkhorn, 1981; Stratford, 1979; Williams 1980; Williams and Fairpo, 1982; Williams and Fairpo, 1984; Bentley, 1994; Quinn and Freeman, 1994; Hunter et al, 1996; and Hunter, 1997). More specifically, recent studies have shown that health visitors could be encouraged to promote early registration and dental attendance of

children (Bentley & Holloway, 1993 and Pine & Deas, 2000). There is, however, no published information to date available on the ability of health visitors to access and carry out caries risk assessment of pre-school children. One of the main aims of this study was to examine the feasibility of such a task.

3.6.2 Recruitment, training and motivation of health visitors

3.6.2.1 Recruitment of health visitors

As noted in the results section 3.5.1, all 57 of the health visitors present at the study outset agreed to participate in the study. Much of this success may be attributed to the approval of the study by the director of nursing services and the clinical nurse managers. Other studies have noted this as an important factor (Bentley et al, 1992). The nurse managers agreed to allow the study team consultation with all the health visitors to describe the study and request participation. They also gave the support necessary for the health visitors throughout the study and were continually informed on the study progress by the study dentist. The clinical nurse managers chaired the NHS Trust monthly meetings for the health visitors and it was imperative the study dentist attended these if matters pertaining to the study were on the agenda. Indeed, these meetings provided a direct communication link with both management and the health visitors when required. The appointment of the liaison health visitor also allowed direct feedback to the study team. At all times the level of health visitor motivation could be monitored and regular monthly meetings with the liaison health visitor maintained contact and any pre-emptive action necessary could be taken to avoid possible health visitor problems.

3.6.2.2 Training of health visitors

The four training sessions organised for the health visitors by the study team were described in 3.4.1.5. These sessions were fundamental to the study and formed the first link in communication between the study dentist and the health visitors. The importance of dental health for pre-school children was already well-accepted by the health visitors and they were keen to see improvements in the levels of decay in children in Scotland. Health visitors are extremely busy health care professionals with high levels of administrative work, in addition to the arduous task of daily home visiting as well as all the other duties expected of them. Many, therefore, expressed dismay at the extra tasks the study protocol would incur but were positive in attitude toward the overall health gain which could be attained if the study was successful and a means of identifying caries risk children could be found.

These training sessions were pivotal to the study in terms of motivation of the health visitors and allowing open discussion to take place. Although opinions were varied and there was some scepticism regarding their ability to commit sufficient time, the health visitors gave support for the study.

Health visitors are an extremely caring profession and it was emphasised at these training sessions that the study team hoped that the project would provide the means by which their valuable role in the community could be recognised and amplified, rather than it being a research task which would offer no benefit to them or the children in the long term. The key message of the study team was a mutual concern for the long term dental health of pre-school children through identification of those

at risk in order to allow implementation of strategies aimed at reducing levels of dental disease.

The health visitors found the technique of saliva sampling easy to carry out at the training sessions and this was supported by the results from the feedback questionnaires during the study (section 3.5.9.2.1, Figure 3.7). The logistics of the provision and collection of sampling kits was fully explored during these sessions and, due to demand, a fourth destination for this purpose was added in the west end of the city. Later in the study, it was requested that an additional fifth site in the east end of the city be made available and this was provided by the study team. It was important to respond to such requests from the health visitors as their co-operation and support was critical to both the implementation and continuation of the study. Much debate was stimulated by circulation of the draft questionnaires. This constructive criticism allowed finalised questionnaires to be formulated and was viewed as a positive sign of the health visitors' willingness to actively participate in the study. The health visitors considered the completion of the questionnaires 'easy' as the results in section 3.5.9.2.1 (Figure 3.8) show. Methods of accessing the children were also explored. Health visitors expressed concerns regarding the ability of the study dentist to attend baby clinics as many of these were held in different health centres on similar days. It was accepted that the study dentist would visit many of the children at home. At the ages of 12-months and 2-years, the child's health visitor carries out developmental screenings. There is no 3-year screening, as this is normally carried out at 3.5 years of age. These screenings provided the opportunity for the health visitor to carry out the caries risk assessment tasks at this

time, if possible. It was suggested at the training sessions that the health visitors may have increased difficulty accessing the children at 3-years of age as a routine visit was not mandatory and many of the children had begun attending nursery schools and playgroups by this age. As many mothers would also have returned to employment by this time, it was proposed by the health visitors that return of the 3-year parental questionnaire may be more problematic. This difficulty in accessing the children at older ages was reflected in the comments provided by the health visitors in the feedback questionnaire (section 3.5.9.2.3, Table 3.9).

The health visitors gave support for the study during these training sessions and the study team was encouraged by the enthusiasm of both the health visitors and the management staff.

As the study progressed, the study dentist trained new health visitors employed by the NHS Trust in Dundee on an individual basis. There were approximately three new health visitors per year and it was necessary to train them on an individual basis to initiate and encourage contact between the study dentist and the health visitor and also to ensure that the proper techniques were adopted.

3.6.2.3 Health visitor motivation

Maintenance of the motivation displayed by the health visitors during the training sessions was constantly pursued by the study team. It was imperative to continually seek health visitor's views, constructive criticism and also ideas on the study progress and future outcomes. One of the main aims of the study was to assess the feasibility of a partnership with health visitors to access the children. To maintain

this partnership required dedication by the study team and also by the health visitors. As previously noted in section 3.4.8, newsletters (Appendix 3.11), buffet lunches and informal meetings were aimed at maintaining health visitor interest. It was also essential that the health visitors felt appreciated and did not perceive themselves as merely tools for research purposes. Continual thanks and praise were given to aid this appreciation. Efforts were made in the correspondences and communications to disseminate findings and offer praise for the continued positive results. A results seminar, which included a lunch, was held following the third year of the study to provide an opportunity for presentation of the results and gain the health visitor's opinion on both the results and their future implications. A final results seminar in June 1999 afforded the study team the opportunity to present the initial study results and praise the health visitors for their hard work and positive attitude.

3.6.3 Consent rates

The consent rates for the four years of the study, grouped together into years 1 and 2 and years 3 and 4 were provided in Table 3.1. As can be seen, the consent rate was maintained in final two years of the study. A number of families refused consent to participate in the study. This reduced from 93 children at the beginning of the study to 92 at its close due to one family moving from the area. A total of 22 children were withdrawn from the study. Most parents or guardians gave no reason for withdrawal. However, the most frequent reason expressed was a difficulty in giving time for the study tasks. Mothers who had returned to work and did not want their child seen in nursery also felt an evening visit was too intrusive on their personal

lives. At the end of the fourth year consent or refusal had never been obtained for a total of 93 children. These children were classed as 'no reply'. For these children, the health visitor was unable to trace the family or the family remained out of contact intentionally or unintentionally. If the family moved from the area, the child was still classed as consented if consent was obtained before moving. If the family moved without consent ever being obtained, these children were not included in the available cohort for consent.

The consent rate for the four years was calculated on the basis of available children for whom written consent forms had been obtained. This included those moved who had initially given written consent. The consent rate for the first two years was 89% and the second two years, also 89%.

The health visitors obtained consent from the parent or guardian of the child at the time of the child's 8 month developmental screening. Subsequent to a request by the health visitors, they were also provided with un-named consent forms for opportunistic contact with the families, some of who found it difficult to attend clinic appointments or did not wish to attend a clinic. The high consent rate achieved was almost certainly due to the commitment of the health visitors. The health visitors in this study were responsible for initial introduction and explanation of the study to the parent or guardian. It has been reported that most new mothers are very receptive to advice and instruction given to them at this stage (Blinkhorn, 1981). Health visitors were, therefore, ideally placed to explain that participation in this study could be of long-term benefit to children in terms of dental health. They were also able to stress the importance of this improved dental health for each

particular family. Results from the health visitor feedback questionnaire (Figures 3.9, 3.10 and 3.11) illustrated that health visitors viewed dental health as a priority and these views may have been transmitted to the parents or guardians of the children at this time. These figures also show that the health visitors regarded dental health as a high priority for the duration of the study. This attitude would have obviously contributed to their ability to obtain consent for 1532 children and collect data for the majority of these children for a 4-year period.

Analysis of the parents or guardians who refused to participate in the study showed most were clustered to specific health visitor numbers (Figure 3.1). This may have been due to the personal attitudes of the health visitors involved or a different system of daily duties. However, Figures 3.3, 3.4, 3.5 and 3.6, the graphs showing the percentage returns of data over the four years of the study, show that health visitor numbers 114 and 108 had, consistently, among the lowest return rates of data. These HV code numbers had 20% and 11% of refusals respectively. It would appear, therefore, that specific health visitors consistently did not participate in the study as readily as their colleagues. This was noted in the comments from some health visitors in the feedback questionnaires (Table 3.9). The study dentist and health visitor liaison person made persistent approaches towards these specific health visitors for the duration of the study, but to no avail. Unfortunately, they did not usually attend feedback seminars or respond to the offer of help in the newsletters.

3.6.4 Pilot study

Of the seven pilot study children, all child saliva samples, health visitor questionnaires and parental questionnaires were returned completed, with no associated difficulties reported by either parents or health visitors. Results of the culture of caries associated microorganisms from the saliva samples showed evidence of bacteria and yeast and provided confirmation that the technique of saliva sampling and the microbiological methodology (see chapter 4.2) was effective. The main study was, therefore, implemented on 1 April 1994.

3.6.5 Dental examination

A dental examination was carried out by the study dentist on a total of 1419 one year olds, 1394 two year olds, 1219 three year olds, and 1365 four year olds (Table 3.2). These numbers represented a percentage of 84%, 83%, 73% and 81% of the total number of consented children at these respective ages. The distribution of the location of the dental examinations altered over the duration of the study. This will be discussed in 3.6.6. Dental examination of the children at 2-years of age was difficult to carry out and more time was required than at 1-, 3- and 4-years with each individual child. Two years of age is a notoriously difficult time in terms of behaviour and attitude. Recent literature has revealed that one in five two year olds has a temper tantrum at least twice a day and one reason for this is frustration at not being able to express themselves fully (Health Education Board for Scotland, 1994). The number of dental examinations was lowest in the third year of the study. This was most likely due to the difficulty in accessing three-year-olds, as the health

visitors had anticipated at the study outset. Many mothers were back at work and these children were distributed around childminders, private nurseries and playgroups. Many of the mothers's had not informed the health visitor of this and much time was spent making appointments for unfruitful home visits. The study dentist was also on maternity leave for a period of 3-months at the beginning of the fourth year which did not allow time for 'catch-up' of those children aged 3-years in the final months of the third year of the study.

3.6.6 Access methods

The overall methods used to gain access to the children for all four years of the study duration were shown in Table 3.3, including the percentage of each access method used in relation to the number of children dentally examined. The 'other' visits included joint and separate visits by the study dentist to locations such as nurseries. However, these have been amalgamated into one 'other' visits category as the number of joint-other visits never exceeded 1% of the total number of children dentally examined and was deemed to be a separate visit to another location by the study dentist. The purpose of the 'other' visit was to analyse the change of location of pre-schoolers as they progress from infants to school age children.

3.6.6.1 Access methods over the four year study duration

Figure 3.2 showed, graphically, the distribution of the access methods used for dental examinations over the four years of the study. It can be clearly seen that separate home visits comprised the most frequent of the visit types for the first three

years. However, in the fourth year, this changed to 'other' visits (separate visit to a location other than home or clinic). This was entirely due to the attendance of the majority of children at nursery school. The number of separate visits (separate home plus separate clinic plus 'other' visits) by the study dentist, i.e. without the health visitor, increased significantly from 59% in the first year to 76% and 75% in years 2 and 3 respectively and again to 88% in year 4. This has implications in terms of the health visitors' ability to work independently. At ages, 1, 2 and 3-years, the study dentist did not attempt to access the children on a separate occasion until the health visitors had completed the study questionnaires. The health visitors, therefore, chose the access method and contacted the study dentist if they wished to carry out a joint home or clinic visit.

At 1-year of age approximately half of the children were accessed jointly and the other half separately. Many of the clinics organised for the 12-month developmental screenings were scheduled on the same day for many of the health centres and it was, therefore, impossible for the study dentist to attend all of these clinics. This may have partly accounted for the number of separate home visits carried out by the study dentist.

The results in Table 3.3 showed the increase in the number of separate visits carried out for dental examination in the second year. This increase could have been due to a number of factors. Firstly, the health visitors had gained confidence with the methodology of data collection and did not require the presence of the study dentist for support. Another factor may have been the nature of the 2-year developmental screening. This requires a prolonged time with a 2-year old to assess mental and

physical development, including speech and motor skills. Health visitors often carry out this assessment at home in a relaxed environment as 2-year olds are notoriously difficult. The health visitor may have felt that the presence of the study dentist, who was unknown to the child, could have had a detrimental effect on the child's behaviour. The increase in the number of visits to other locations may be explained by the return of parents to employment. At age 3-years there was a significant increase in the number of children accessed at other locations. This figure increased from less than half of one percent (4 children) at age 1-year to 5% (75 children) at age 2-years, followed by another increase to 13% (157 children) at age 3-years. This increase may be explained by an increased number of children attending nursery, playgroup and childminders as mothers return to employment. Tayside Regional Council nurseries do not accept children until 3.5 years of age in Dundee, therefore, most of these children attended local playgroups run by social services or church groups or private nurseries. Results show, however, that the health visitors continued to be successful at accessing the children for caries risk assessment data independent from the study dentist. A number of health visitors continued to prefer joint visits and the majority of these were carried out in a clinic environment. This was similar to the results for the second year. The methods used to gain access to children at age 4-years for dental examination differed dramatically from the other years of the study. In this year, the study dentist did access the children prior to completion of questionnaires by the health visitor. The steep rise in the number of children accessed in nursery schools in the fourth year is not surprising as most children have a pre-school year in nursery in Scotland. One of the important results

from this fourth year was the fact that only 12% of the children were accessed jointly by the study dentist and health visitors. This shows an undoubted ability of health visitors to access pre-school children for completion of data relating to the caries risk status of a pre-school child. The results also show that the health visitors felt confident about carrying out these procedures by themselves.

3.6.6.2 Summary of access methods

The results of analysis of the methods of access have many potential implications. As previously noted in this chapter, health visitors are in a unique position in the community to gain access to pre-school children and in the past this has been utilised in terms of oral health care for the prevention of oral disease (Fuller et al, 1992), and increased dental registration (Bentley et al, 1993 and Pine and Deas 2000). No previously published study, however, has compared differences in the methods used to access pre-school children for collection of caries risk assessment data. This study has shown that, at the ages of 1-, 2-, 3- and 4-years, health visitors accessed large numbers of children for caries risk assessment independently from the study dentist and that the percentage of separate visits significantly increased at ages 2-, 3- and 4-years compared to age 1-year ($p<0.001$). For those children accessed jointly, the majority were seen in a clinic environment and as the children increased in age a significantly larger proportion were accessed in environments such as playgroups, nurseries and childminders. These results provide clear indications of the methods which might be used to successfully access pre-school children at different ages.

Results of percentage returns of saliva samples and questionnaires by the health visitors (Tables 3.4 – 3.6) indicated the high capability of the health visitors to collect caries risk assessment data. This capability, when combined with the ability of the health visitors to successfully access the children, whether at home, at a clinic or some other location, has many implications for future identification of those children at highest risk of developing dental caries. Identification of such 'at risk' children could allow implementation of preventive strategies for these children and so provide the opportunity to reduce the levels of decay in 5-year olds and assist achievement of the target set for 5-year olds in The Oral Health Strategy for Scotland document (1995) and Public Health White Paper - Towards a Healthier Scotland (1999).

3.6.7 Microbiological saliva sampling

Looking at the 4-year duration of the study, 1436 saliva samples were obtained by the health visitors from the children at age 1-year, 1381 at age 2-years, 1247 at age 3-years and 1150 at age 4-years (Table 3.4). These represent percentage returns of saliva samples of 85%, 82%, 74% and 68%, respectively, of the total numbers of children consented at these ages. The saliva samples were obtained by the health visitors using the system of provision and collection of sampling kits described briefly in section 3.4.5 and in more detail in Chapter 4.2. No studies published to date have shown results for the ability of health visitors to collect saliva samples from pre-school children. The return rates for the saliva samples in this study were high and this can be attributed to the dedication of the health visitors.

Results from the methods used to access the children (Figure 3.2) showed that the health visitors frequently carried out this saliva sampling independently of the study dentist and increasingly so as the study progressed. The health visitors showed some concern with regard to obtaining enough saliva from the children, as it was often difficult to ensure the child's mouth was open long enough to allow the sampling loop to be drawn across the child's tongue. Results from the microbiological saliva sampling, however, (Appendix 6) showed that bacteria were indeed cultured from the saliva samples and reassurance was given to the health visitors. These results indicated that the health visitors were capable of carrying out microbiological saliva sampling of pre-school children as part of a caries risk assessment.

3.6.8 Questionnaires

3.6.8.1 Health visitor questionnaire (HQ)

The health visitors completed health visitor questionnaires in their own time. Data from the child's medical records was required and this was often time consuming for the health visitor to obtain. As shown in Table 3.5, the number of these questionnaires was 1426 in year-1, 1394 in year-2, 1261 in year-3 and 1163 in year-4. This represented 85%, 83%, 75% and 69% of the consented cohort, respectively, at these ages. The high number of these questionnaires returned mirrored the commitment of the health visitors throughout the duration of the study period.

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Chapter 2.2.9 and the predictive capability of this hunch factor will be discussed in Chapter 6.

3.6.9 Distribution of saliva sample and questionnaire returns by health visitors

Figures 3.3, 3.4, 3.5 and 3.6 show the percentage return of child saliva samples, health visitor questionnaires and parental questionnaires for each of the four years of the study respectively. These return rates show some differences in participation between the various health visitors in Dundee. It can also be noted that the same group of health visitors (code numbers 108, 112, 114) consistently found problems with data collection and were in the bottom five for return rates, although these were a minority of the total health visitor group. Discussion of these return rates with the health visitors by the liaison health visitor and the study dentist revealed problems with workload and organisation of the individual health visitors. None of the health visitors had encountered specific problems with the study methodology, other than finding the time to follow it. The results from the health visitor questionnaire showed that finding time for the data collection was fairly difficult. However, the majority of the health visitors, to their credit, were able to complete data collection. As noted in section 3.6, however, there were specific health visitors who did not, for whatever reason, fully participate in the study and measures to encourage increased participation of health visitor codes 114 and 122/127 were unsuccessful. This can be highlighted by following the changes for the health visitor with code number 122, who changed to number 127. This health visitor code number 127 developed

problems in the final two years, following a previously successful two years. This new HV 127 was health visitor number 122, who then did not collect data as number 127. Interestingly, the new health visitor 122 did not appear to encounter the same problems in the final two years.

3.6.10 Health visitor feedback questionnaire (HVFQ)

3.6.10.1 Return rates

The return rate of these questionnaires for the second, third and fourth years of the study were shown in Table 3.8. These rates were comparable to those of other studies involving health visitors (Bentley et al, 1992). The first feedback questionnaire was issued in the second year of the study. This was due to the time commitment required in the first year of the study for set-up of the administrative aspects, including computer databases. The percentage returns of 75%, 71% and 64% were not as high as was hoped by the study team. However, it was viewed as an encouraging sign, as the health visitors would have been expected to be more likely to inform the study team of any problems encountered using the feedback questionnaire. The results from the questions were also encouraging.

3.6.10.2 Results from questions

Figures 3.7 – 3.11 and Table 3.9 show the results from the questions asked of the health visitors in the feedback questionnaire. These results reflected the overall satisfaction the health visitors had with the study methodology and their opinions on various aspects of the study.

3.6.10.2.1 Study methodology

3.6.10.2.1.1 Saliva sampling procedure

Figure 3.7 showed the results of the questions related to the sampling procedure. This graph suggests that the majority of health visitors were able to use the sampling kit without any great difficulty. The number of health visitors who found the technique 'very easy' reduced incrementally over the 3 years. This was most likely related to the increased awareness of the children and their increased capability of saying 'no'.

3.6.10.2.1.2 Questionnaires

Figure 3.8 showed the results of the question related to completion of the health visitor questionnaire. This graph most clearly shows that the health visitors had no difficulty completing the health visitor questionnaire and did not find the questions confusing.

3.6.10.2.2 Dental health as a priority for health visitors

The question, "on a scale of 1 to 10 how do you rate dental health as a priority?" was asked in each of the three feedback questionnaires. Figures 3.9, 3.10 and 3.11 showed the results of this question for each of the three years. These results show that, as mentioned in section 3.6.3, the majority of health visitors found dental health to be a priority for the duration of the study. For each of the three years respectively, 89%, 73% and 91% rated it a priority of 6 or greater out of 10, with 28%, 22% and 36% rating it a priority of 10 out of 10. Only 4%, 2% and 3% rated it

a priority of less than 5 out of 10. It should be emphasised that this positive attitude toward dental health from a profession, which has such close contact with mothers, and children from before birth until school age should be actively encouraged and utilised.

3.6.10.2.3 Additional comments from health visitors

An open question requesting any other comments was asked for each of the three issues of the feedback questionnaire. Some of the answers to these questions were given in Table 3.9. Many of these comments prompted light-hearted responses from the health visitors such as, "no way am I sticking my fingers in the mouth of a two-year old!" These convivial responses reflected a good-humoured response to the study in general and were assessed by the study team as a positive response. Other, more formal, responses regarding problems with accessing children were recorded and assistance offered in general terms in the following newsletters to the health visitors. As noted in section 3.4.7 the questionnaire was distributed anonymously. This allowed the health visitors unrestricted freedom of speech. It was an encouragement to the study team that no individual health visitor voiced the view that the study was not worthwhile or could not succeed.

3.6.10.2.4 Conclusions from health visitor feedback questionnaire (HVFQ)

In conclusion, the majority of health visitors found it relatively easy to participate in the study, access the children and collect caries risk assessment data. They viewed

dental health as being a priority and responded with humour and constructive criticism to an open question for comments.

3.7 Conclusions of chapter

1. The design of the study supported collection of caries risk assessment data for pre-school children at ages 1-, 2-, 3- and 4-years.
2. Health visitors in Dundee were successfully recruited to participate in this 4-year study to access pre-school children for caries risk assessment.
3. Health visitors obtained consent for 89% of an available cohort of 1681 pre-school children to participate in the study. This consent rate was maintained for the study duration.
4. Health visitors accessed and obtained caries risk assessment data (microbiological saliva samples, health visitor questionnaires and parental questionnaires) in partnership with a study dentist for over 68% of the consented children at ages 1-, 2-, 3- and 4-years, a minimum number of 1149 children each year.
5. Health visitors accessed a greater number of pre-school children separately for caries risk assessment at ages 2- and 3-years compared to age 1-year and the greatest number separately at age 4-years. This has implications for future targeting of pre-school children.
6. A health visitor feedback questionnaire demonstrated that the majority of health visitors had no difficulty participating in the study.
7. The majority of health visitors in Dundee viewed dental health as a priority.

3.8 Hypotheses of chapter tested

Hypothesis 3.1

That health visitors in Dundee can be ***recruited*** *to participate in a 4-year longitudinal study to identify in a large cohort of pre-school children those at risk of developing dental caries.*

All the health visitors agreed to participate in the study and continued to collect data for the 4- year duration.

Thus the hypothesis is proved.

Hypothesis 3.2

That health visitors can gain consent for a longitudinal caries risk assessment study of pre-school children.

Health visitors gained consent for 1683 from a total of 1890 children born and resident in Dundee between 1 April 1993 and 31 March 1994. This represented a consent rate of 89%. At the close of the study, the consent rate was still a remarkable 89%

Thus the hypothesis is proved

Hypothesis 3.3

That it is feasible to employ existing health visitors to collect caries risk assessment data (involving microbiological saliva sampling and questionnaire completion) for the majority of a large cohort of pre-school children for a 4-year longitudinal study.

All the health visitors for the city of Dundee obtained 1436, 1381, 1247 and 1150 saliva samples, 1426, 1394, 1261 and 1163 health visitor questionnaires and 1405, 1342, 1250 and 1149 parental questionnaires for the children at ages 1-, 2-, 3- and 4-years of age respectively. In response to a feedback questionnaire, the health visitors did not encounter any great difficulties with the collection of this data and were able to fit these tasks into their daily duties.

Thus the hypothesis is proved.

Chapter 4: Materials and methods

4.1 Dental examination

A dental examination was carried out on consented pre-school children at 1-, 2-, 3- and 4-years of age. The examination used a combination of direct vision and illumination by a penlight (Ross Promotional Products), with the child in the supine position at age 1-year (Appendix 4.1) and upright position at ages 2-, 3- and 4-years. The results of the dental examination were immediately recorded on to a dental examination data form (Appendix 4.2). Examination was carried out as close to the child's birthday as was feasible at ages 1, 2 and 3-years and within one month of saliva sampling by the health visitor. A study carried out by Schroder and Granath (1983) found that dietary habits and gingival status registered in 3-year-olds could be considered representative for a period of time preceding this age. A period of one month either side of the child's birthday and as close as possible to saliva sampling by the health visitor was, therefore, considered an acceptable time for the dental examination. At the time of dental examination at ages 2, 3 and 4-years the study dentist assessed the oral cleanliness of the mouth, defined as the presence or absence of plaque. Recent reports from Scottish epidemiological surveys have found that the presence of plaque was associated with high levels of decay in 5-year olds (Pitts et al, 1996 and Pitts et al, 1998). However, this thesis has focused on data collected at age 1-year for caries at age 4-years and therefore, this variable was not included in the analysis.

At ages 1, 2 and 3-years, the trigger for dental examination by the study dentist (HBM) was receipt of the parental and / or health visitor questionnaire to the administration centre (see section 4.3). If dental examination had not taken place at the same time as saliva sampling and questionnaire completion by the child's health visitor, upon receipt of either one of the study questionnaires, an appointment was made by HBM to visit the child at home for dental examination. In the fourth year of the study, many of the children were dentally examined in nursery schools (see chapter 3.5.5).

4.1.1 Caries diagnostic methods

4.1.1.1 Introduction

A review of the methods of caries diagnosis is outwith the scope of this thesis and the reader is referred to the many excellent existing reviews (e.g. Pitts, 1991(a); Pitts, 1991(b); Pitts, 1992; Longbottom, 1992; Lussi, 1993; Angmar-Mansson and Ten Bosch, 1993 Stookey, 1996; and Verdonschot et al, 1999). The aim of this section is to describe the diagnostic methods used in this study and give the reader an insight into the reasons for their choice. In a review, Ismail (1997) stated that the prevention of dental caries today, and in the next century, must be based on appropriate detection of dental caries in its earliest stages. He stated that we should not only detect "cavities" but also early signs of demineralisation and disease activity. In this paper, he synthesised the current literature on the validity and reliability of clinical diagnosis of pre-cavitated carious lesions, made recommendations for clinical diagnosis of pre-cavitated lesions and identified areas

for further research. In a more recent presentation Ismail presented a review of the clinical diagnostic criteria of early childhood caries (ECC). This aimed to provide information for a workshop convened by the National Institute of Dental and Craniofacial Research (NIDCR) to develop diagnostic criteria for ECC and case definitions for S-ECC ("severe early childhood caries") (Ismail, 1999). Prior to set-up of the study described in this thesis, the research protocol included diagnosis of non-cavitated enamel caries as well as more advanced dentinal and pulpal lesions. This was in order to: 1) identify risk factors associated with caries initiation; 2) to allow the possibility of secondary caries prevention in future targeting; 3) monitor enamel caries activity / progression and 4) relate d_1 caries to d_3 caries in terms of caries risk.

4.1.1.2 Diagnostic methodology

The caries status of each child examined was diagnosed at the $d_1$caries into enamel threshold (Appendix 1.1). This level of diagnosis included non-cavitated enamel lesions in addition to dentinal lesions and those involving the pulp. All lesions were recorded according to the following criteria developed for the Dundee Selective Threshold methods for caries detection / diagnosis (Fyffe, 1996).

U Unerupted

The tooth was unerupted, or congenitally absent, or missing for reasons unknown.

6 Missing due to caries

Teeth were regarded as missing due to caries if they had been extracted because they were carious. Teeth, which were absent for any other reason were not included in this category.

T *Missing due to trauma*

A tooth missing which could be directly related to a specific traumatic experience.

X *Excluded*

A tooth surface for which a judgement could not be formed.

G *Present and sound*

No visual signs of treated or untreated dental caries. Partially erupted teeth were given the benefit of the doubt and scored G, unless there was cause for concern on the visible surfaces.

W *White spot lesion*

An intact surface with no clinically detectable loss of substance with a white or cream coloured area of increased opacity presumed carious by the examiner.

B *Brown spot lesion*

An intact surface, no clinically detectable loss of substance, with a brown / black discolouration, presumed to be carious by the examiner.

E *Enamel cavity*

A lesion with demonstrable loss of surface but no visual, clinical evidence of the lesion penetrating dentine.

D *Dentine lesion (non-C)*

A carious lesion into dentine but no visible evidence of cavitation.

C *Dentine cavity*

Surfaces were regarded as falling into this category if there was a carious cavity into dentine.

A Arrested dentinal decay

Caries in dentine, which in the opinion of the examiner, was no longer active.

P Pulpal involvement

A carious cavity which involved the pulp, necessitating an extraction or pulp treatment.

F Filled, no decay

Surfaces which contained a satisfactory permanent restoration of any material.

5 Filled and decay

A restored surfaces contiguous with enamel, dentinal or pulpal decay.

R Filled, needs replacing (no decay)

A filled surface which required replacement i.e. was extensively chipped or cracked or was causing damage to adjacent structures. Lesions or cavities which contained a temporary dressing, or cavities from which a filling had been lost were included unless there was also evidence of caries.

$ Sealed surface

A surface which contained some type of fissure sealant

4.1.2 Training and calibration of the study dentist (HBM)

The study dentist was trained and calibrated by a dentist (CL) experienced in caries diagnostic methodology and previously trained according to the criteria above. Training was carried using extracted deciduous teeth. Over a three-day period, both

HBM and CL repeatedly examined previously extracted deciduous teeth until all types of lesions in the diagnostic criteria could be reliably identified and recorded. Final calibration was carried out on simulated quadrants. These quadrants were constructed using a deciduous canine, deciduous first and deciduous second molar embedded in alginate impression material. This technique aimed to simulate the intra-oral tooth/gingival interface. Five sets of four quadrants each containing three teeth gave a total of sixty teeth for calibration. These simulated mouth were examined by HBM and CL using the caries diagnostic methodology described and the results recorded on the dental examination data form. The data recorded in the forms was subsequently entered onto SPSS software and kappa scores evaluated.

4.1.3 Reproducibility of the caries diagnostic methodology

4.1.3.1 In vitro reproducibility

4.1.3.1.1 Year 1, 2 and 3 of study

The five sets of four quadrants of deciduous teeth described above were used to evaluate intra-examiner variability. All 60 teeth were examined using the caries diagnostic methodology described and results recorded onto dental examination data forms. The teeth were subsequently examined following a period of one week and the kappa scores evaluated.

4.1.3.1.2 Year 4 of study

During the fourth year of the study, due to a period of maternity leave by HBM, a second examiner (JP) was employed for a three-month period. This examiner

carried out one hundred dental examinations of 4-year-olds following prior calibration according to the criteria outlined in 4.1.2. In summary, HBM and JP and JP and CL examined all 60 teeth and results were recorded onto dental examination forms. Examination was repeated one week later and kappa scores evaluated for both inter- and intra-examiner reproducibility.

4.1.3.2 In vivo reproducibility

Following advice from the study statistician, fifty-seven children aged 3-years were re-examined by the study dentist (HBM) in the third year of the study. These children were selected 'blind' to HBM on a random basis by CL and the parents contacted directly by HBM to arrange a second dental examination. This examination was carried out within one month of the initial examination. Identical caries diagnostic methodology was employed and results recorded onto the dental examination data form. These results were analysed by SPSS computer software and kappa scores evaluated for intra-examiner reproducibility.

4.2 Microbiological methodology

4.2.1 Bacteriological saliva sampling

4.2.1.1 Sampling procedure

It was imperative that the technique used for sampling the oral flora in this study was applicable to the sample size, age of the subjects, easily used by the health visitors (and study dentist when required) and reflected the numbers and species of oral flora present. The tongue-loop method (Beighton, 1986) fulfilled these

requirements and was used for the duration of the study. A literature review on the methodological considerations was provided in chapter 2.2.4.2. The tongue-loop technique used an individually wrapped sterile and disposable, plastic 10 microlitre loop (Mackay and Lynn Ltd.) The loop was drawn across the dorsal surface of the tongue until filled with saliva and tongue debris to obtain a 10 microlitre saliva sample. The loop was then immediately agitated into a 1.2ml vial (Camlab Laboratories) containing 1.0ml of fastidious anaerobic broth (FAB) (Lab M) to completely dislodge all tongue material from the loop into the vial. The loop and wrapper were then disposed into ordinary household waste. The vial was placed into an insulated styrofoam storage box (Whatman Scientific Ltd.) and this box placed into a specially adapted cool-bag (Jencons Scientific Limited) for transport. Instructions for the saliva sampling technique were clearly printed on the upper surface of each styrofoam box to aid consistent methodology (Appendix 4.3). The cool-bags were silver in colour with wooden handles and consisted of two layers of polyethylene with an intermediate layer of polyester wadding and a wooden carrying handle. They required modification, which involved a reduction in length followed by heat sealing. An experienced laboratory technician carried out this process in the Dental Hospital / School. The cool-bags were easily identified by health visitors and unique in purpose for this study. The cool-bag and its contents was termed a 'sampling kit' and consisted of styrofoam storage box containing sampling vial; sampling loop; spare label to identify vial and pentel-pen (Appendix 4.4)

4.2.1.2 Sample identification

Starting in March 1994, a sampling pack was sent to every health visitor each month until the end of the study. This contained: a covering letter; a list of the study children due for sampling the following month; a set of three labels showing the identification (study) number of each child; a parental and health visitor questionnaire for each child; return addressed envelopes for completed questionnaires; a reminder letter for the parent/guardian of each child and envelopes and stamps for correspondence with the parent/guardian. One label was used for each of the parental and health visitor questionnaires and the sampling vial. The study number used on these labels was identical to that used for the duration of the study and ensured confidentiality. As noted previously, each cool-bag contained a spare label and pentel-pen in case they were required by the health visitor at the time of sampling.

4.2.1.3 Parental sampling

During the first year of the study, using the technique described in 4.2.1, a saliva sample was taken by the study dentist (HBM) from the study child's mother to assess the level of cariogenic microorganisms. This was to investigate the potential of this factor as a predictor of decay in the child. The procedure was carried out at the time of dental examination of the child. Verbal consent from the mother was obtained by HBM at the time of sampling, although previous written consent had been obtained at the start of the study. The sampling vial was labelled with the child's study number followed by 'M'.

4.2.2 Storage and transport of saliva samples

Subsequent to saliva sampling and placement of the sample in the cool-bag, the health visitor put the cool-bag as quickly as possible into a cool, insulated igloo located in one of five designated sites within Dundee. These locations were namely, Broughty Ferry Health Centre, Douglas Clinic, Ryehill Health Centre, Wallacetown Health Centre and Westgate Health Centre. The locations were selected with the agreement of the health visitors as they represented a reasonable distribution across the city of Dundee for the convenience of the health visitors. These five locations were used for both provision of 'fresh' sampling kits and collection of 'used' ones following sampling. In each centre there were two igloos clearly labelled 'fresh' and 'used' and the name of the centre (Appendix 4.5). Each evening between 16:00 and 17:00 hours these igloos were exchanged with two replacement igloos. The first of these contained the fresh sampling kits with ice packs to maintain coolness ('fresh' igloo) and the second contained ice packs only, in preparation to receive the saliva samples obtained the following day. For the initial 2-years of the study this daily replacement system was carried out by members of the study team and thereafter by means of a taxi service. The taxi picked up the ten igloos, two for each health centre, from the Dental School at 16:00 hours daily and followed the same route each day. This ensured that the health visitors could be given approximate times when fresh sampling kits were available for the next day and the latest times saliva samples taken that day could be placed in 'used' igloo at each health centre. The health visitors could pick up fresh sampling kits at any time but if the kit was not being used that day it required refrigeration at -

5°C. Refrigerated sampling kits could be kept for up to one week before use, but as soon as the saliva sample was taken it was imperative that it was returned to a 'used' igloo that day and as soon as possible after sampling. All health visitors were advised that no saliva samples were to be kept overnight as the bacteria would not remain viable. Saliva samples taken after pick-up times were collected directly by the study dentist or delivered to the Dental Hospital by the health visitor. The study dentist was contactable at all times by mobile telephone, the number of which was issued to all health visitors and repeated in any correspondence.

4.2.3 Preparation of saliva sampling kits

Preparation of saliva sampling kits for the 'fresh' igloos was undertaken on a daily basis and the igloos were prepared for collection by the taxis at 15:45 hours. The cool-bags contained 1 sterile sampling loop, a spare set of labels, pen and the styrofoam storage box containing the sampling vial. 'Fresh' igloos contained three ice packs and eight cool-bags (sampling kits) and 'used' igloos three ice packs only. The ice-packs were kept in the freezer compartment of a Dental School refrigerator after collection each day. If requested, additional cool-bags were provided for health visitors.

4.2.4 Strategies to help maintain bacterial viability

The styrofoam boxes used to store the samples were designed by the manufacturers to provide insulation and protection of the sampling vials and these boxes were placed directly into the cool-bags. These cool-bags were manufactured from two

layers of polyethylene with an intermediate layer of polyester wadding and a wooden carrying handle. Their design provided insulation of the styrofoam box. Manufactured in one size only, the cool-bags required reduction by approximately one half followed by heat sealing.

Following saliva sampling the cool-bag was placed in an igloo by the health visitor in one of the five locations in the city. These igloos were insulated plastic containers, kept cool with several sealed ice packs, and systematically renewed daily to ensure a low temperature was maintained. All igloos were returned to the microbiological laboratory in the Dental Hospital by 17:15 hours which, since HVs did not sample before 09:15, ensured a time lag of no more than 8 hours had elapsed between the time of saliva sampling by the health visitor and microbiological processing.

4.2.5 Microbiological processing

4.2.5.1 Preparation of saliva samples

Subsequent to delivery of the saliva samples to the microbiological laboratory, all sample identification numbers, date of collection and a separate chronological sample number for each were carefully noted in a logbook (Appendix 4.6). This information was subsequently transferred to computer database at a later date. Each sample was dispersed by vortexing for 10 seconds with a miximatic rotamixer (Jencons Scientific Limited).

4.2.5.2 Plating out of saliva samples

Following dispersion, 0.1ml (100µl) of sample fluid from the sample vial was pipetted onto each of 3 selective media plates, namely, rogosa agar (ROG), bacitracin in mitis salivarius agar (BMSA) and sabouraud's dextrose agar (SAB) (all media purchased from Oxoid/Unipath Ltd). These plates had been clearly labelled with the correct chronological sample number using a water resistant marker pen. The sample fluid was then spread over each media plate using a disposable L-shaped spreader (Lab M).

4.2.5.3 Bacteriological media

All media was purchased as either partial or complete formulations. They were reconstituted from the dehydrated state according to manufacturer's instructions and sterilised if required in 250 ml volumes by autoclaving at a temperature of 120°C for twenty minutes. All media was prepared, poured and dried in the microbiological laboratory using a laminar flow cabinet (MDH Ltd) and drying cabinet. They were subsequently stored in a commercial refrigerator at <8^{0}C for a maximum period of one week before use.

4.2.5.3.1 Fastidious anaerobic broth (FAB) (Lab M)

FAB was used as the transport medium for the microorganisms in the saliva samples. 29.7g of powder was weighed, dispersed in 1 litre of distilled water and agitated for 10 minutes. After bringing to the boil while gently mixing it was then dispersed into 250ml polypropylene bottles leaving minimal headspace. Following

autoclaving and cooling, 1.0ml of the broth was pipetted into 1.2ml Nunc cryotubes (CamLab Ltd) and these were stored in the refrigerator until required for the sampling kits.

4.2.5.3.2 Sabouraud's dextrose agar (SAB)

This selective medium was used to recover yeast species as described by Odds (1988).

65g of powder was suspended in 1 litre of distilled water (Elga Ltd.) and brought to the boil until dissolved completely. It was then sterilised by autoclaving and placed in a water bath at 50^{0}C. After cooling, media plates were poured directly and stored in the refrigerator.

4.2.5.3.3 Rogosa agar (ROG)

This selective medium was used to isolate species of lactobacilli as described by Rogosa, Mitchell and Wiseman in 1951.

82g of powder was suspended in 1 litre of distilled (Elga Ltd.) water and brought to the boil until dissolved completely. 1.32ml of glacial acetic acid (Sigma Chemical Co.) was added and mixed thoroughly. The mixture was heated to 90 - 100^{0}C for 2 to 3 minutes with frequent agitation, then allowed to cool. No autoclaving was required and after cooling, media plates were poured directly and stored in the refrigerator

4.2.5.3.4 Bacitracin in mitis salivarius agar (BMSA)

This selective medium was used to recover mutans streptococci (*streptococcus mutans* and *streptococcus sobrinus*) using the technique as originally described by Gold, Jordan and Van Houte in 1973.

90g of powder was suspended in 1 litre of distilled water (Elga Ltd.) and 150g of sucrose per litre was added (Sigma Chemical Co.). This mixture was heated to boiling until dissolved completely, then sterilised by autoclaving and allowed to cool to 50 - 55^{0}C. 1ml of filter sterilised potassium tellurite solution (2ml potassium tellurite ampoule in 5ml of distilled water) per litre was added (Sigma Chemical Co.) A bacitracin solution of 20 units per ml was made up to give 100ml (Sigma Chemical Co.). This was filter sterilised and 10ml per litre added. The potassium tellurite solution was made from 2ml stock solution bought in glass ampoules. The solution was made up to 7ml by adding 5ml of de-ionised water. The final solution was filtered through a sterile 0.2µl cellulose acetate filter directly into a sterile vessel and this solution was stored at < 8^{0}C until required.

The bacitracin stock solution was prepared by dissolving 2000 units of bacitracin in 100ml of de-ionised water and sterilised as for potassium tellurite.

The media plates were poured, labelled and stored in the refrigerator.

4.2.5.4 Incubation of media plates

Following plating out of the saliva samples the plates were cultured by incubation at 37^{0}C for 72 hours. BMSA and ROG plates were taped together using autoclave tape and placed in an anaerobic jar (Don Whitley Scientific Ltd and Philip Harris

Scientific). The anaerobic atmosphere was maintained using anaerobic gas generating kits (Unipath Ltd). These were replaced each time the jar was opened for addition or removal of plated samples. After taping, the SAB plates were placed directly on the incubator shelf and cultured aerobically for an identical time period.

4.2.5.5 Identification of caries associated microorganisms

Following the required incubation period, the plates were examined directly for colonies of caries associated microorganisms. The lowest detection level was 10^3 Colony Forming Units (CFU) per ml of saliva. CFU's were counted and examined for appearance, colour, odour and structure. Representative colonies from the plates were tested using the catalase test (Sigma Chemical Co.) (Appendix 4.7) and stained for microscopic examination using Gram's stain (Pro-Lab Diagnostics) (Gillies and Dodds, 1984) (Appendix 4.8). All results were recorded on a microbiological data sheet (Appendix 4.9) and entered into computer database at a convenient time.

4.2.5.5.1 Presumptive identification of mutans streptococci

Numbers of mutans streptococci were provisionally identified on the basis of their unique colony morphology on BMSA agar. Appearance consisted of a 1mm, dark blue, crenated, raised raspberry shaped colony which was embedded in the media and difficult to remove. Gram stain revealed gram positive cocci in chains. Achievement of a gram stain was unpredictable, however, due to the resilient nature of the colonies. *Streptococcus mutans* and *streptococcus sobrinus* colonies were differentiated by virtue of the latter being surrounded by a halo on the media. Both

species responded negatively to the catalase test. A photograph of a representative media plate has been provided (Appendix 4.10).

4.2.5.5.2 Presumptive identification of lactobacilli species.

This bacterial species isolated on ROG agar revealed a colony morphology comprising white or cream colour domes of varying size (1-5mm) which had a creamy texture. Gram stain revealed gram positive rods of varying size microscopically. These colonies responded negatively to the catalase test. A photograph of a representative media plate has been provided (Appendix 4.11).

4.2.5.5.3 Presumptive identification of yeast species.

Species of yeast on SAB agar were large (2-10mm), white colonies with a matt appearance. They were creamy and had a distinctive 'brewer's yeast' odour. Gram stain revealed gram positive large ovals with budding hyphae. The colonies responded positively to the catalase test. A photograph of a representative media plate has been provided (Appendix 4.12).

4.2.6 Confirmatory identification of caries associated microorganisms

It was imperative that bacterial and yeast colonies presumed to be different on the basis of their colony morphology were adequately characterised by biochemical or serological techniques (de Soet et al, 1987). Therefore, to confirm presumptive identification, representative colonies were delivered to Professor David Beighton's laboratory at King's College School of Medicine in London. This was achieved in

two ways. Firstly, a representative batch of isolation plates were packed and sent by courier post at regular intervals as soon as feasibly possible after examination in Dundee. These were examined again directly in the microbiological laboratory in London and re-cultured, if possible, for further testing.

The second method of obtaining confirmatory identification was using 'protect vials' (Lab M). These protect vials contained plastic beads within a liquid support medium. Presumptively identified colonies were removed carefully from the media using the sterile loop provided. This was agitated into the 'protect vial' to allow adherence of the colony to the beads contained within the vial. The vial was inverted gently 16 times and excess liquid siphoned with a 1ml sterile plastic Pasteur pipette (Mackay and Lynn Ltd). The vial was stored in an insulated styrofoam storage box in a - 80^0C freezer until transportation to London. To allow same day delivery and reduce the risk of damage, the study dentist transported the boxes which contained the vials directly from Dundee to London. All isolation plates were sent to London for the first 3 months of data collection and subsequently reduced to batches at intervals of 6 months for the remainder of the study period. Preparation of 'protect vials' was continued for the entire duration of the study period.

4.2.6.1 Confirmatory identification from 'protect vials'

The colonies contained in the 'protect vials' were cultured on media plates and definitive identification in London carried out in two ways. Firstly, a repeated direct visual examination of the colonies on the media plate was carried out and recorded to allow comparison with results obtained in Dundee. Secondly, a series of

biochemical tests were carried out. Sugar fermentation tests were used for mutans streptococci colonies (Beighton et al, 1991) and enzyme substrate tests for lactobacilli species.

4.2.6.1.1 Sugar fermentation tests for mutans streptococci

Colonies of mutans streptococci were incubated in Todd Hewitt broth for 48 hours. 135microl. of sugar was added to sterile microlitre trays (corning cell wells) (LIP Equipment & Services Ltd) with a control row at the end. On addition of 45µl of the incubated broth, the trays were incubated anaerobically at 37^0C for 24 hours. Yellow indicated a positive result for the well, purple negative. For arginine hydrolysis, 45µl of Nessler's reagent was added to the well. Orange indicated a positive result. All results were noted in chart like form (Appendix 4.13) and copies held in Dundee.

4.2.6.1.2 Enzyme substrate tests for lactobacilli species.

Non - sterile microlitre trays (Medicell International Ltd.) were used for this test. Colonies were removed directly from the isolation plate with a sterile cotton swab. These were suspended in 1ml of buffer and 20 µl of enzyme was placed into each well followed by 45 µl of each isolate and incubated at 37^0C for 3 hours. Substrate hydrolysis was determined by measuring fluorescence on the Perkin Elmer Fluorimeter. Production of enzyme hydrolysis was positive if an increase in fluorescence of 5 units above the control value was present. This was viewed on an ultra-violet light box.

4.2.7 Validation of microbiological methodology

4.2.7.1 Sampling procedure

The tongue-loop method was chosen for its ease of use and simplicity. The study dentist (HBM) followed the guidelines provided in the methodological paper by Beighton (1986) and carried out the procedure on ten subjects. This procedure was repeated one week later. Following consistent results, HBM trained all health visitors in the technique of tongue-loop sampling during a series of training sessions (chapter 3.5.1.5). A pilot study (chapter 3.5.7) involved saliva sampling of 7 children by their health visitors, who experienced no problems with the sampling technique.

4.2.7.2 Identification of caries associated microorganisms

The study dentist (HBM) and study technician (VW) were responsible for the microbiological processing procedures. Full training of both HBM and VW was carried out in London supervised by Professor David Beighton. A copy of the training sessions have been provided (Appendix 4.14). To validate these procedures a certain number of microbiological plates were analysed repeatedly for the presence of mutans streptococci, lactobacilli and yeast by both HBM and VW (inter-examiner reproducibility). Plates chosen at random were read twice by VW to assess intra-examiner reproducibility. Plates were also sent for confimatory identification to London (see section 4.2.9)

These results will be provided in chapter 5.2.3

4.2.7.3 Laboratory procedures

Guidelines for the laboratory methodology, which included preparation of sampling kits (section 4.2.3), preparation of saliva samples (section 4.2.5.1), plating out of saliva samples (section 4.2.5.2), media preparation, use, and storage (section 4.2.5.3), incubation of media plates (section 4.2.5.4) and identification of micro-organisms (section 4.2.5.5) were provided in a Standard Operating Procedures (SOP) booklet (Appendix 4.15). This ensured that both HBM and VW followed an identical procedure when carrying out these techniques.

4.3 Socio-demographic and health behaviour data

Socio-demographic and health behaviour data was collected by means of study questionnaires.

4.3.1 Parental questionnaire (PQ)

The parental questionnaire (Appendix 3.6) was given to the parent / guardian of the study child by the health visitor at the time of saliva sampling (section 4.2). This questionnaire was completed annually for the 4-year duration of the study. The questions contained within this questionnaire provided data on: breast/bottle feeding; meals; drinks; snacks; toothbrushing; fluoride supplementation; child-care and food shopping. Some alterations were made to the questionnaire in the fourth year of the study such as elimination of the question on breast-feeding and addition of a question regarding the mother's education.

4.3.2 Health visitor questionnaire (HQ)

The health visitor questionnaire (Appendix 3.7) was completed by the study child's health visitor either at the same time as saliva sampling or at a later date. Again, a questionnaire was completed for each of the four years of the study. Much of the data required was contained within the child's medical records but the health visitors needed to ask the parent or guardian the answers to a few questions. The health visitor questionnaire provided data on: medical development such as weight, height and head circumference; immunisation status; ethnic origin; illnesses; medication; weaning; use of comforter; vitamin supplementation; feeding problems; family history; parental employment status; parental health; parental smoking and housing status. The questionnaire became shorter in the second year as developmental details became less frequent. A question requesting an updated address was added from the second year onward.

Questionnaires for each child in any given month were sent to each appropriate health visitor approximately one month in advance of the due risk assessment date (that is, the child's birthday). A covering letter, list of children for that month and three labels were also enclosed in the pack, termed the sampling pack, with two envelopes, one for return of the questionnaires and another for correspondence by the health visitor with the parent or guardian. The ten digit study number of the child was hand written on each of the three labels and designated for use on the saliva sampling vial, parental questionnaire and health visitor questionnaire.

4.3.3 Collection of health visitor's hunch data

In each of the four years of the study the health visitor was asked a specific question which was emphasised on the health visitor questionnaire. This question asked was, "Is the child at high risk of developing dental decay? Yes/No". As described by Disney et al (1992), this hunch reflected the examiner's subjective personal judgement or "gut feeling" about whether the child was at risk. There was no training given with regard to this question. The health visitor had to decide the answer on the basis of their knowledge of the child and the child's environment.

4.3.4 Development and reproducibility of study questionnaires

The study questionnaires were finalised following consultation with the study statistician and all the health visitors prior to implementation of the study protocol. No problems were encountered with the study questionnaires in the pilot study (section 3.4.2)

The questionnaires were validated using fifty repeated questionnaires completed within the field setting. The study children were chosen at random and the data entered onto SPSS database. Results of this reproducibility are provided in chapter 5.3.2.

4.4 Statistical analysis

4.4.1 Introduction to statistical analysis

A review of the methods of analysis in caries risk assessment was provided in chapter 2.2.2. This chapter describes the statistical methodology used for data analysis in this thesis.

4.4.2 Correlation matrix

A correlation matrix technique was used (SPSS) in order to sieve the vast amount of data collected and provide the most significant factors, collected at age 1-year, associated with caries in the children at age 4-years (Appendix 5.3). Once these factors were identified, they could be analysed with respect to their ability to predict caries.

4.4.3 Logistic regression analysis

Logistic regression analysis for 'any risk' and 'high risk' model development was carried out on the data both at the d_1 and d_3 levels of caries diagnosis to ascertain the predictive capability of factors at age 1-year associated with caries at age 4-years. Results of the logistic regression analysis have been provided in chapter 5.4.2.

4.4.4 Chi-squared Automatic Interaction Detector Analysis (CHAID)

This novel method of analysis was carried out on SPSS computer software. This comprised a tree-based classification system derived by Kass (1980), shown to improve upon traditional approaches (Magidson, 1988) and enhanced by Statistical Innovations (SPSS/PC + CHAID). This was a software package designed to flexibly generate prediction trees from non-parametric data. Data on the children at 1-year of

age was entered into the analysis until the best combination of predictors was established for caries at 4-years of age. The data collected from the question on the health visitors opinion of risk ('hunch') in year-1 was not complete, however (1026 answered but 400 did not). Since it was considered that this factor might be a significant predictor it was decided that, rather than exclude one third of the data set, it would be reasonable to use the HV opinion at age 2-years (n = 169) where the data was missing at 1-year. It was thought unlikely that the HV opinion would alter between these two years (analysis of HV opinion of risk for the children at age 1- and 2-years showed acceptable reproducibility (kappa = 0.5)). All other data contained in the analysis was collected in year-1. The factors from fully completed child data sets were entered into the analyses to obtain the best predictors (284 child data sets). The non-predictive factors were then excluded and the CHAID analysis applied to the 506 child data sets for which complete data was available for all these best predictors. This resulted in a new set of predictors for this larger data set. The process was then repeated - exclusion of non-predictive factors and application of CHAID analysis – to the 784 child data sets ('high risk') and 697 data sets ('any risk') for which complete data sets were available for that new set of best predictors. This analysis created branching groups until the predetermined critical value of probability and/or cell size were reached. The predetermined critical value was the prevalence of disease in the data set to which the CHAID analysis was applied - i.e. in the primary cell. The predetermined maximum cell size was ten. Ends of branches were labelled as high or low risk depending on the prevalence of disease within that data set relative to that in the primary cell. A higher relative prevalence

designated a 'high risk' label. Sensitivities and specificities were calculated by generation of two-by-two tables of predicted versus actual disease (see chapter 2.2.2.1.1 and Table 2.1). This analysis was carried out for $d_1mft > 0$, $d_3mft > 0$, $d_1mft \geq 3$ and $d_3mft \geq 3$. The results have been provided in chapter 5.4.3.

4.4.5 Risk model development

In order to develop a caries risk model for those 4-year olds at 'any risk' of caries and those at 'high risk' of caries, a method of obtaining a cut-off point for the high risk children was required. To achieve this the distribution of disease within the population was examined (Tables 5.3 and 5.4 and Figures 5.1 and 5.2) and the nearest point to which the relatively smallest proportion of the population had the relatively largest proportion of disease was identified. Consideration was also given to the fact that recent reports have confirmed that in many communities 80% of dental caries occurs in 20% of the population (Whelton and O'Mullane, 1998). For the purposes of this study, therefore, a d_1mft a value of equal to or greater than 3 for each child was classified as 'high risk'. At this d_1mft value 27.4% of the population had 82.4% of the disease at 4-years and was the most appropriate value for detection of the minority of children with the majority of the disease. At the d_3mft threshold of diagnosis, a value of equal to or greater than 3 missing, filled or decayed teeth was selected as the cut-off point for 'high caries risk'. At this level of detection, 15.5% of the population had 78.5% of the disease at 4-years.

These $d_1$mft and $d_3$mft levels for risk assessment were then analysed using logistic regression analysis and CHAID analysis to provide an 'any risk' and a 'high risk' prediction model for caries in 4-year olds using data collected at age 1-year.

These results will be provided in chapter 5.4.

Chapter 5: Results

5.1 Dental examination

The number of dental examinations carried out was shown in Table 3.2 and is given in Table 5.1. To briefly summarise the total number of children examined at ages 1, 2, 3 and 4 was 1419, 1394, 1219 and 1365 respectively.

5.1.1 Caries prevalence

5.1.1.1 Caries prevalence at the d_1 threshold of diagnosis

The results of the caries prevalence data at the d_1 level of caries diagnostic threshold are shown in table 5.1.

Table 5.1: Number of dental examinations, caries prevalence and percentage of children with decay at ages 1-, 2-, 3- and 4-years at the d_1 threshold of caries diagnosis.

	Number of Dental Examinations	**Number of Children with Decay**	**Percentage of Children with Decay**
Year 1	1419	39	3%
Year 2	1394	172	12%
Year 3	1219	321	27%
Year 4	1365	674	49%

5.1.1.2 Caries prevalence at the d_3 threshold of diagnosis

The results of the caries prevalence data at the d_3 level of caries diagnostic threshold are shown in table 5.2.

Table 5.2: Number of dental examinations, caries prevalence and percentage of children with decay at ages 1-, 2-, 3- and 4-years at the d_3 threshold of caries diagnosis.

	Number of Dental Examinations	**Number of Children with Decay**	**Percentage of Children with Decay**
Year 1	1419	6	0.4%
Year 2	1394	57	4%
Year 3	1219	144	12%
Year 4	1365	449	33%

5.1.1.3 Caries distribution within the population at age 4-years

Tables 5.3 and 5.4 show the distribution of caries within the population of 4-year-olds in the study at the $d_1$mft and $d_3$mft thresholds of diagnosis respectively. Figure 5.1 shows the caries distribution of those children dentally examined at age 4-years and Figure 5.2 the distribution of disease within those children with caries at age 4-years.

Table 5.3: Distribution of caries at the $d_1$mft threshold of diagnosis at age 4-years (1365 subjects, 3213 $d_1$mft teeth)

$d_1$mft	No.	% population	% disease	Complement Population	Complement disease
0	691	50.6	0.0	49.4	100.0
1	104	58.2	3.2	41.8	96.8
2	128	67.6	11.2	32.4	88.8
3	68	72.6	17.6	27.4	82.4
4	95	79.6	29.4	20.4	70.6
5	44	82.8	36.2	17.2	63.8
6	58	87.0	47.1	13.0	52.9
7	41	90.0	56.0	10.0	44.0
8	45	93.3	67.2	6.7	32.8
9	24	95.1	73.9	4.9	26.1
10	18	96.4	79.5	3.6	20.5
11	3	96.6	80.5	3.4	19.5
12	18	97.9	87.3	2.1	12.7
13	9	98.6	90.9	1.4	9.1
14	12	99.5	96.1	0.5	3.9
15		99.5	96.1	0.5	3.9
16	4	99.8	98.1	0.2	1.9
17		99.8	98.1	0.2	1.9
18		99.8	98.1	0.2	1.9
19		99.8	98.1	0.2	1.9
20	3	100.0	100.0	0.0	0.0

Table 5.4: Distribution of caries at the $d_3$mft threshold of diagnosis at age 4-years (1365 subjects, 1917 $d_3$mft teeth)

$d_3$mft	**No.**	**% population**	**% disease**	**Complement Population**	**Complement disease**
0	916	67.1	0.0	32.9	100.0
1	115	75.5	6.0	24.5	94.0
2	72	80.8	13.5	19.2	86.5
3	51	84.5	21.5	15.5	78.5
4	53	88.4	32.6	11.6	67.4
5	28	90.5	39.9	9.5	60.1
6	32	92.8	49.9	7.2	50.1
7	22	94.4	57.9	5.6	42.1
8	22	96.0	67.1	4.0	32.9
9	11	96.8	72.2	3.2	27.8
10	12	97.7	78.5	2.3	21.5
11	6	98.2	82.0	1.8	18.0
12	11	99.0	88.8	1.0	11.2
13	3	99.2	90.9	0.8	9.1
14	6	99.6	95.3	0.4	4.7
15	1	99.7	96.0	0.3	4.0
16	1	99.8	96.9	0.2	3.1
17		99.8	96.9	0.2	3.1
18		99.8	96.9	0.2	3.1
19		99.8	96.9	0.2	3.1
20	3	100.0	100.0	0.0	0.0

Figure 5.1: Distribution of disease ($d_1$mft and $d_3$mft) within those children dentally examined at age 4-years.

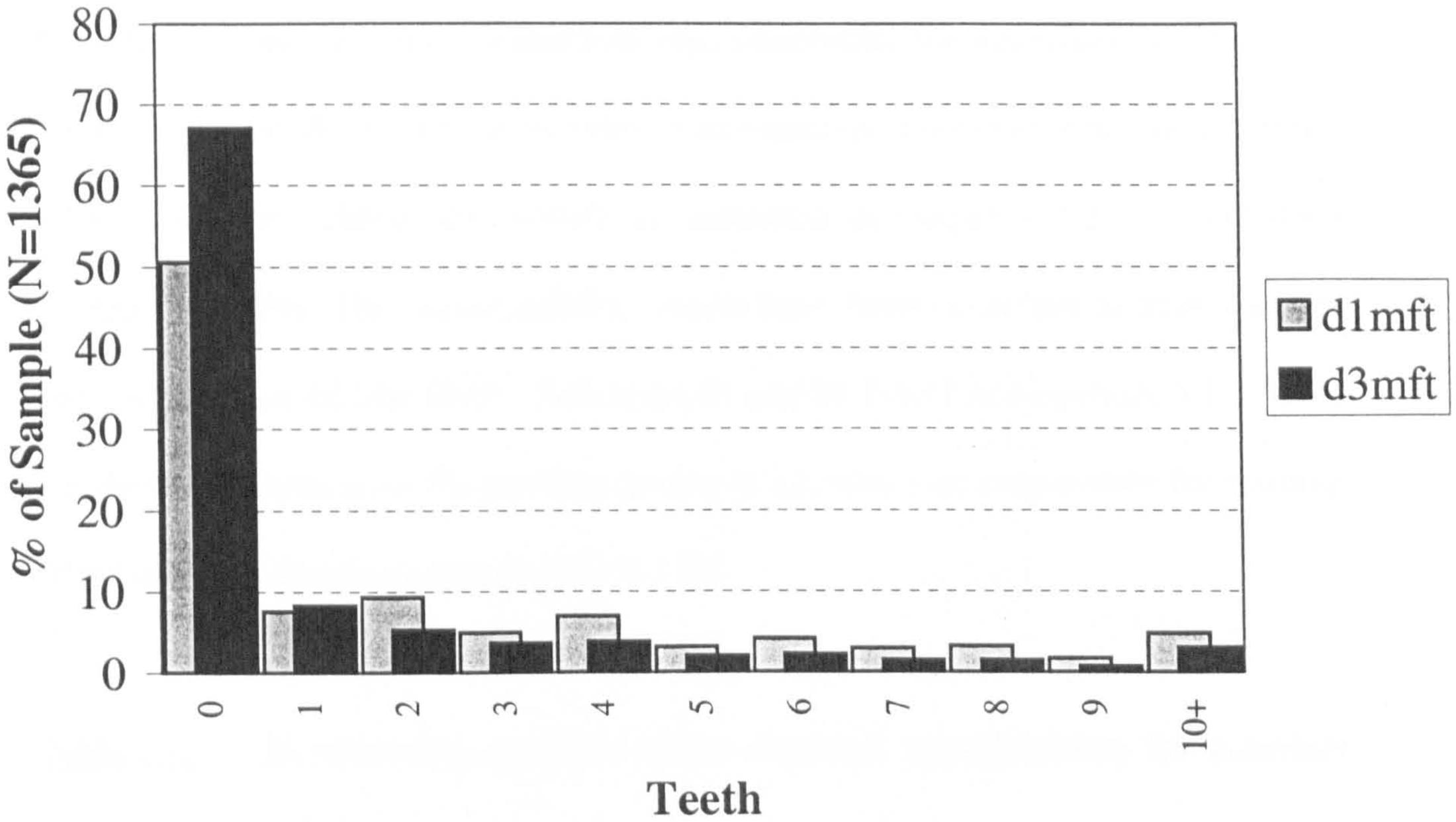
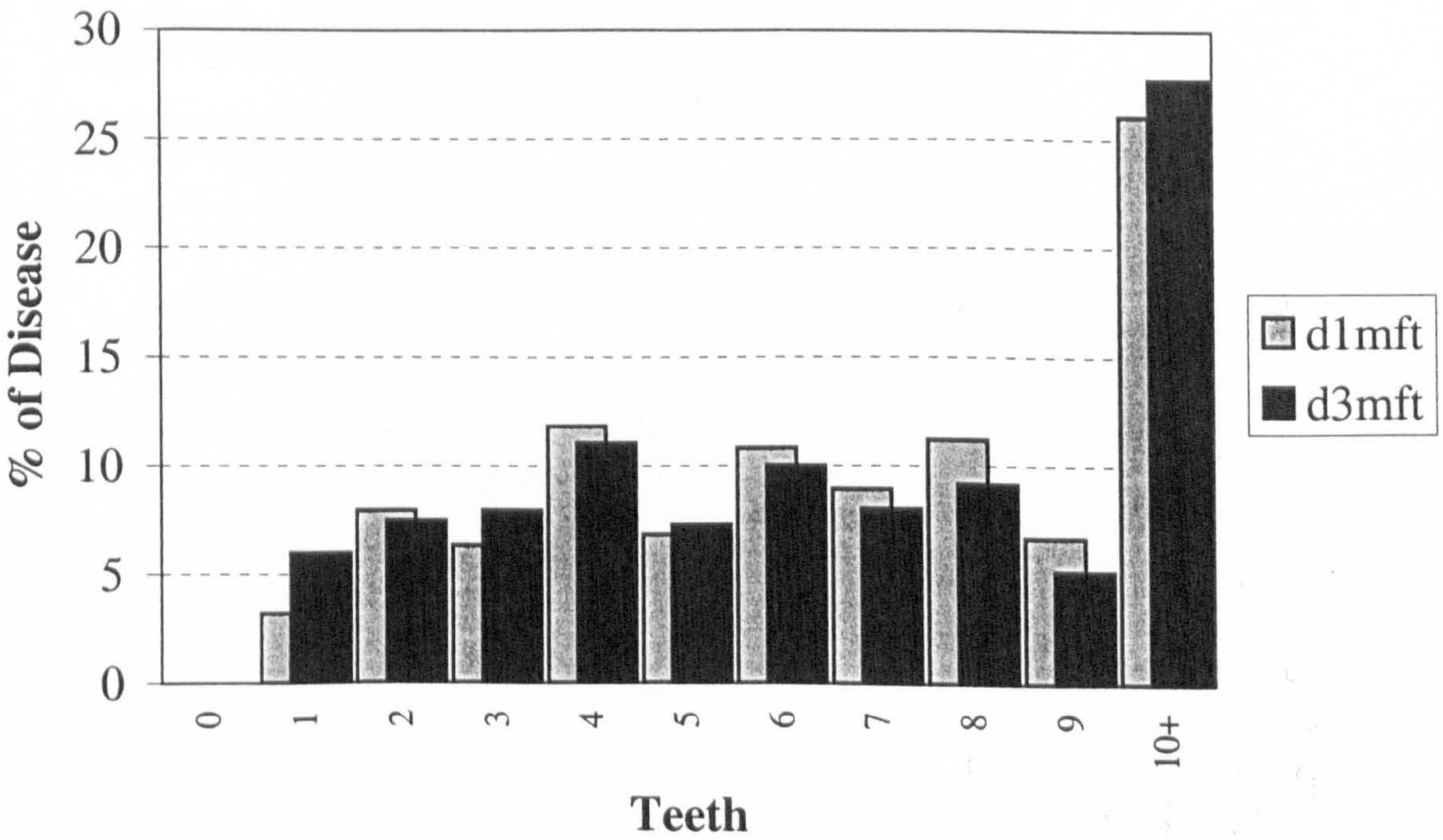


Figure 5.2: Distribution of disease ($d_1$mft and $d_3$mft) within the study population with caries at age 4-years.



5.1.1.4 Caries diagnosis reproducibility

5.1.1.4.1 In vitro reproducibility

5.1.1.4.1.1 In vitro intra-examiner reproducibility for examiner HBM

Table 5.5 shows the results of in vitro intra-examiner reproducibility for examiner HBM using the calibrations models as described in chapter 4.1.2. (5 calibration models, 60 teeth) The reproducibility results have been presented as kappa values and displayed in tabular form. Full analysis can be found in Appendix 5.1. Table 5.6 shows calibration to the training dentist (CL), who was responsible for training HBM in caries diagnosis (see chapter 4.1.2)

Table 5.5: In vitro intra-examiner caries diagnosis reproducibility for examiner HBM.

HBM v HBM	1994	1996	1997
Kappa value ($d_1$mft)	0.62	0.79	0.96
Kappa value ($d_3$mft)	0.89	0.96	1.0

Table 5.6: Calibration reproducibility for examiner HBM v CL in year-1 of study

HBM v CL	
Kappa value ($d_1$mft)	0.92
Kappa value ($d_3$mft)	1.0

5.1.1.4.1.2 In vitro intra-examiner reproducibility for examiner JP in year-4 of study

In the fourth year a second examiner (JP) was calibrated to carry out dental examinations (see chapter 4.1.3.1.2). Reproducibility (Kappa values) of this calibration is shown in table 5.7.

Table 5.7: In vitro intra-examiner caries diagnosis reproducibility in year-4 for examiner JP.

JP v JP	Year 4
Kappa value ($d_1$mft)	0.69
Kappa value ($d_3$mft)	0.81

5.1.1.4.1.3 In vitro inter-examiner reproducibility for examiners HBM and JP in year 4 of study

In the fourth year of the study in vitro inter-examiner reproducibility was carried out on the original calibration models, as a result of the introduction of a second examiner (JP) for a short period. These results are shown in Table 5.8.

Table 5.8: In vitro inter-examiner caries diagnosis reproducibility in year-4 of study for examiners HBM v JP and CL v JP (calibration).

HBM v JP	Year 4
Kappa value ($d_1$mft)	0.37
Kappa value ($d_3$mft)	0.61
CL v JP	
Kappa value ($d_1$mft)	0.71
Kappa value ($d_3$mft)	0.65

5.1.1.4.2 In vivo reproducibility

5.1.1.4.2.1 In vivo intra-examiner reproducibility for examiner HBM

In vivo reproducibility was carried out on a total of 57 children chosen at random by CL. These children were examined by the study dentist (HBM) as normal and then re-examined following an arranged appointment one week later. The results of these repeat examinations are shown in Table 5.9.

Table 5.9 In vivo intra-examiner caries diagnosis reproducibility for examiner HBM.

HBM v HBM	57 children re-examined
Kappa value ($d_1$mft)	0.70
Kappa value ($d_3$mft)	0.67

5.1.1.4.2.2 In vivo inter-examiner reproducibility for examiners HBM and JP in year-4 of study

In the fourth year of the study examiners HBM and JP examined the same children at different times. This, however, was done at varying time intervals ranging from one week to 4 months and, therefore, it was possible that changes could have occurred in the mouths of the children in the intervening time. Table 5.10 shows the results of these analyses.

Table 5.10: In vivo caries inter-examiner diagnosis reproducibility for examiners HBM and JP.

HBM v JP	10 children re-examined
Kappa value ($d_1$mft)	0.75
Kappa value ($d_3$mft)	0.66

5.2 Microbiological saliva sampling

5.2.1 Number of saliva samples obtained from study children

The number of saliva samples obtained in the study was shown in Table 3.4. To summarise, the number of samples obtained at ages 1, 2, 3 and 4-years was 1436, 1381, 1247 and 1150 respectively.

5.2.2 Number of saliva samples obtained from mothers of the study children

The total number of saliva samples obtained from the mothers was 1170.

5.2.3 Reproducibility of microbiological methodology

5.2.3.1 Intra-examiner reproducibility

5.2.3.1.1 Intra-examiner reproducibility for identification of mutans streptococci

Table 5.11 shows the kappa scores for forty, randomly selected repeat saliva sample analyses by the study technician (VW)) for the identification of mutans streptococci. Data analysis (cross tabulations) have been provided in Appendix 5.2.

Table 5.11: Intra-examiner reproducibility of mutans streptococci identification by the study technician (VW).

VW v VW	Forty plated samples
Kappa value	1.0

5.2.3.1.2 Intra-examiner reproducibility for identification of lactobacilli species.

Table 5.12 shows the results of the kappa values for the identification of lactobacilli species by the study technician (VW). For these purposes thirty randomly selected saliva samples were plated out and re-read to test intra-examiner reproducibility

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Table 5.12: Intra-examiner reproducibility of lactobacilli species identification by the study technician (VW).

VW v VW	Thirty plated samples
Kappa value	1.0

5.2.3.1.3 Intra-examiner reproducibility for identification of yeast species

Table 5.13 shows the results of the kappa values for the identification of yeast species by the study technician (VW). For these purposes thirty randomly selected saliva samples were plated out and re-read to test intra-examiner reproducibility.

Table 5.13: Intra-examiner reproducibility of yeast species identification by the study technician (VW).

VW v VW	Thirty plated samples
Kappa value	0.87

5.2.3.2 Inter-examiner reproducibility

Inter-examiner reproducibility between the study technician (VW) and the study dentist (HBM) was carried out using twenty randomly selected plated saliva samples.

5.2.3.2.1 Inter-examiner reproducibility for identification of mutans streptococci

Table 5.14 shows the kappa scores for twenty randomly repeated saliva sample analyses by HBM and VW for the identification of mutans streptococci.

Table 5.14: Inter-examiner reproducibility of mutans streptococci identification by the study technician (VW) and study dentist (HBM).

HBM v VW	Twenty plated samples
Kappa value	1.0

5.2.3.2.2 Inter-examiner reproducibility for identification of lactobacilli species.

Table 5.15 shows the results of the kappa values for the identification of lactobacilli species by the study technician (VW) and study dentist (HBM). For these purposes twenty randomly selected saliva samples were plated out and re-read to test inter-examiner reproducibility.

Table 5.15: Inter-examiner reproducibility of lactobacilli species identification by the study technician (VW) and study dentist (HBM).

HBM v VW	Twenty plated samples
Kappa value	1.0

5.2.3.2.3 Inter-examiner reproducibility for identification of yeast species

Table 5.16 shows the results of the kappa values for the identification of yeast species by the study technician (VW) and study dentist (HBM). For these purposes twenty randomly selected saliva samples were plated out and re-read to test inter-examiner reproducibility.

Table 5.16: Inter-examiner reproducibility of yeast species identification by the study technician (VW) and study dentist (HBM).

HBM v VW	Twenty plated samples
Kappa value	1.0

5.3 Socio-demographic and health behaviour data

5.3.1 Number of completed questionnaires

These results were provided in chapter 3.5. To summarise, 1405, 1342, 1250 and 1149 parental questionnaires and 1426, 1394, 1261 and 1163 health visitor questionnaires were completed for the first, second, third and fourth years of the study respectively.

It must be noted, however, that not all questionnaires were fully completed and risk assessment analysis was carried out on completed data sets. 891 health visitor and 822 parental questionnaires were completed for the same study children at ages 1, 2, 3 and 4-years.

5.3.2 Reproducibility of study questionnaires

The study questionnaires were validated by randomly repeated completion of fifty questionnaires.

Table 5.17 shows the reproducibility results (kappa values) for repeated questions from the parental questionnaires for the 4-year study duration.

Table 5.17: Reproducibility of completed questions in the parental questionnaire (PQ) for the 4-year study duration.

Question	Kappa value
Was your child bottle fed?	0.55
Was your child breast fed?	0.77
Does your child have supper?	0.75
Does your child have a snack?	0.74
Does your child have a biscuit as a snack?	0.77
How do you do your shopping?	0.75
Are your child's teeth brushed?	0.62
By child?	0.67
By parent?	1.0

Table 5.18 shows the reproducibility results (kappa values) for repeated questions from the health visitor questionnaires (HQ) for the 4-year study duration.

Table 5.18: Reproducibility of completed questions in the health visitor questionnaire (HQ) for the 4-year study duration.

Question	Kappa value
Immunisation status?	1.0
Age at weaning?	0.46
Breast feeding?	1.0
Mother's employment?	0.68
Mother's marital status?	0.72
Number of siblings?	0.56
Mother's smoking status?	1.0
Father's employment?	1.0

5.4 Results of caries risk assessment analysis

5.4.1 Correlation matrix

The results of the correlation matrix analysis has been provided in Appendix 5.3.

5.4.2 Logistic regression analysis

For completeness, the results of the full logistic regression analysis have been provided in Appendix 5.4 and these results will be outlined in the following sections. The final logistic analysis was based on the results from data obtained for 697 1-year old children at d_1 and d_3 mft > 0 and 784 children at d_1 and $d_3 \geq 3$. These numbers reflect the complete data sets available for these children at these disease thresholds.

5.4.2.1 Factors at age 1-year for caries at age 4-years

5.4.2.1.1 $d_1$mft at age 4-years > 0 – 'any caries risk' model

Table 5.19 shows the sensitivity and specificity values for the logistic regression analysis for $d_1$mft > 0 at age 4-years. The most predictive factors were: DEPCAT, HV opinion of risk, parental smoking, use of a dummy and food or drink at night, according to the formula – Risk = 2.91 (constant) + 0.35 (DEPCAT) – 0.70 (HV op) – 0.49 (Psmoke) – 0.41 (dummy) – 0.51 (f/dnight).

Table 5.19: Sensitivity and specificity values for prediction at age 1-year of $d_1$mft > 0 at age 4-years using logistic regression analysis (n = 697).

$d_1$mft > 0	
Sensitivity	54%
Specificity	70%

5.4.2.1.2 $d_3$mft at age 4-years > 0 – 'any caries risk' model

Table 5.20 shows the sensitivity and specificity values for the logistic regression analysis for $d_3$mft > 0 at age 4-years. The most predictive factors were: DEPCAT, HV opinion of risk, parental smoking, and food or drink at night according to the formula – Risk = 2.07 (constant) + 0.39 (DEPCAT) – 0.79 (HV op) – 0.69 (Psmoke) –0.51 (f/dnight).

Table 5.20 Sensitivity and specificity values for prediction at age 1-year of $d_3mft > 0$ at age 4-years using logistic regression analysis (n = 697).

$d_3mft > 0$	
Sensitivity	26%
Specificity	89%

5.4.2.1.3 d_1mft at age 4-years ≥ 3 – 'high caries risk' model

Table 5.21 shows the sensitivity and specificity values for the logistic regression analysis for $d_1mft \geq 3$ at age 4-years. The most predictive factors were: HV opinion of risk, use of feeder cup, parental smoking and housing type according to the formula – Risk = - 0.66 (constant) – 0.56 (HV op) + 0.52 (Feeder cup) – 0.34 (Psmoke) + 0.68 (housing).

Table 5.21 Sensitivity and specificity values for prediction at age 1-year of $d_1mft \geq 3$ at age 4-years using logistic regression analysis (n = 784).

$d_1mft \geq 3$	
Sensitivity	16%
Specificity	95%

5.4.2.1.4 d_3mft at age 4-years ≥ 3 – 'high caries risk' model

Table 5.22 shows the sensitivity and specificity values for the logistic regression analysis for $d_3mft \geq 3$ at age 4-years. The most predictive factors were: HV opinion of risk, use of feeder cup, housing type and snacking according to the formula – Risk = - 1.41 (constant) – 0.69 (HV op) + 0.54 (Feeder cup) + 0.78 (housing) - 0.73 (snack).

Table 5.22: Sensitivity and specificity values for prediction at age 1-year of $d_3mft \geq 3$ at age 4-years using logistic regression analysis.

$d_3mft \geq 3$	
Sensitivity	0%
Specificity	100%

5.4.3 Chi-squared Automatic Interaction Detector analysis (CHAID)

The complete CHAID analyses have been provided in Appendix 5.5. The definitive models chosen for each category have been provided in the following sections, accompanied by the sensitivity and specificity values for each model. The methodology for CHAID analysis was fully described in chapter 4.4.4.

5.4.3.1 Factors at age 1-year for caries at age 4-years

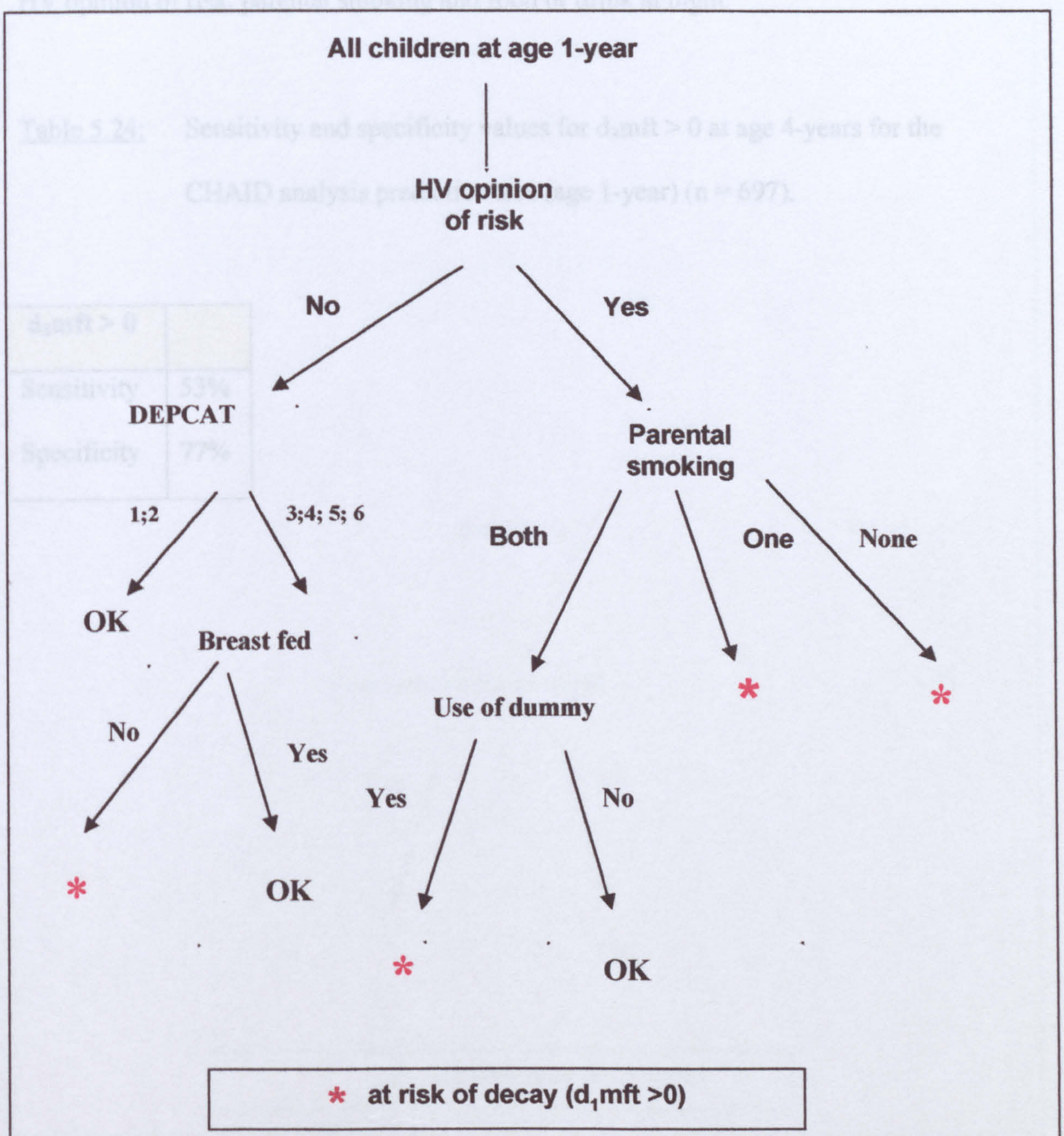
5.4.3.1.1 d_1mft at age 4-years > 0 – 'any caries risk' model

The diagrammatic tree diagram of this risk assessment is shown in figure 5.3 and the sensitivity and specificity values in Table 5.23. The most predictive factors were: HV opinion of risk, DEPCAT, parental smoking, breast feeding and use of a dummy.

Table 5.23: Sensitivity and specificity values for $d_1mft > 0$ at age 4-years for the CHAID analysis prediction tree (age 1-year) (n = 697).

$d_1mft > 0$	
Sensitivity	67%
Specificity	57%

Figure 5.3: Diagrammatic CHAID risk assessment model for prediction at age 1-year of $d_1mft > 0$ at age 4-years (Se = 67%, Sp = 57%) (n = 697)



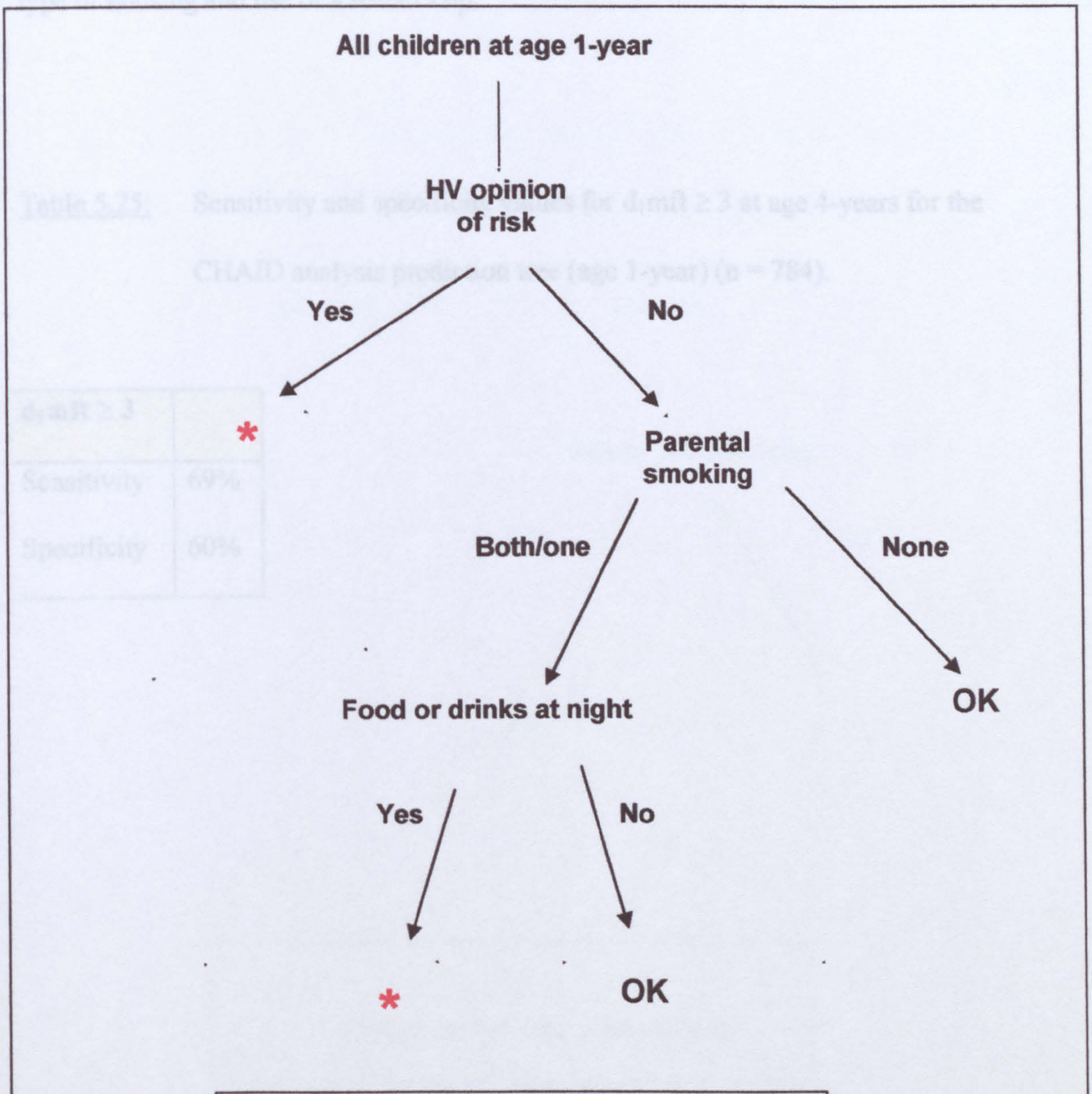
5.4.3.1.2 d_3mft at age 4-years > 0 – 'any caries risk' model

The diagrammatic tree diagram of this risk assessment is shown in figure 5.4 and the sensitivity and specificity values in Table 5.24. The most predictive factors were: HV opinion of risk, parental smoking and food or drink at night.

Table 5.24: Sensitivity and specificity values for $d_3mft > 0$ at age 4-years for the CHAID analysis prediction tree (age 1-year) (n = 697).

$d_3mft > 0$	
Sensitivity	53%
Specificity	77%

Figure 5.4 Diagrammatic CHAID risk assessment model for prediction at age 1-year of $d_3mft > 0$ at age 4-years (Se = 53%, Sp = 77%) (n = 697).



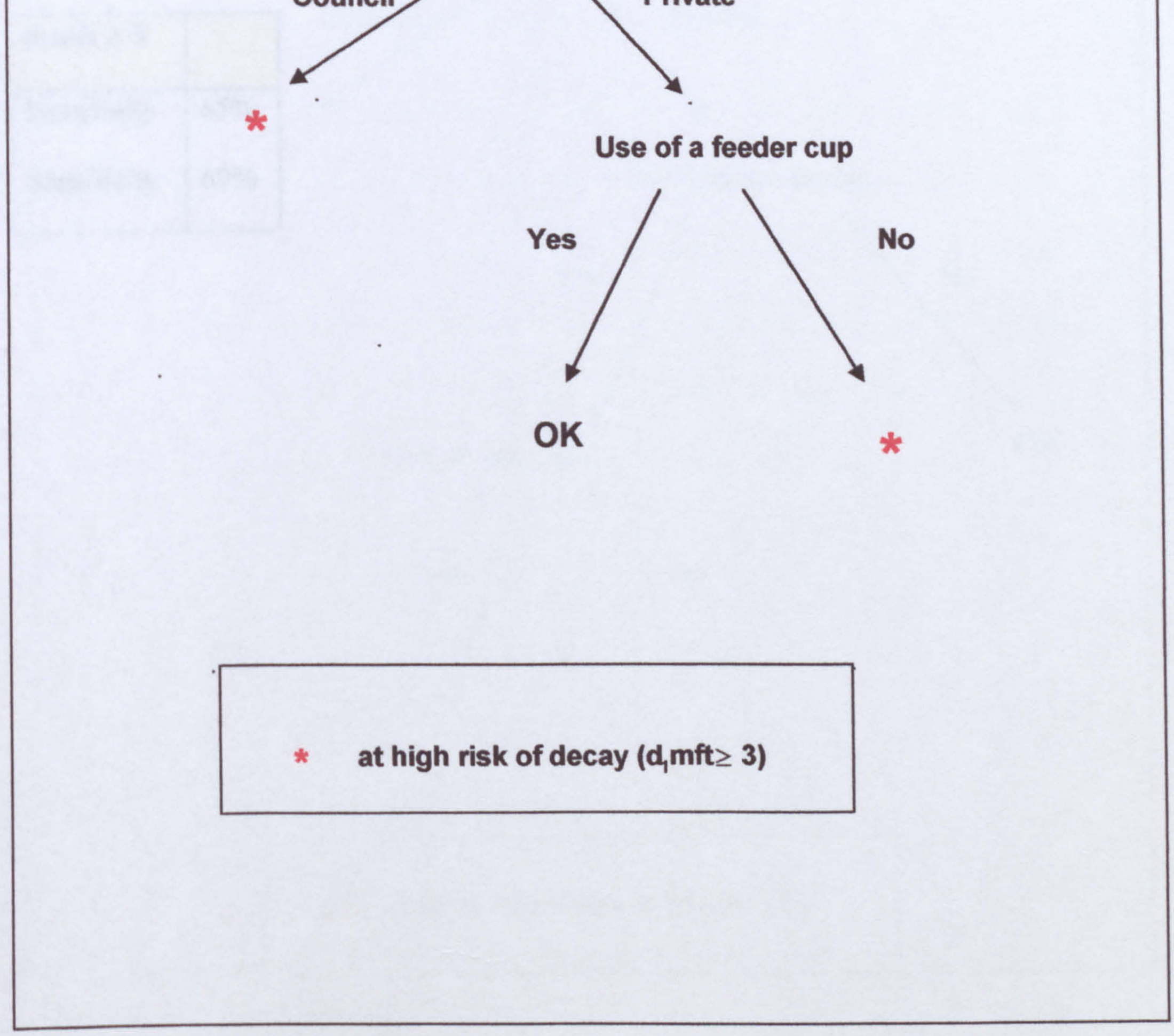
5.4.3.1.3 $d_1$mft at age 4-years ≥3 – 'high caries risk' model

The diagrammatic tree diagram of this risk assessment is shown in figure 5.5 and the sensitivity and specificity values in Table 5.25. The most predictive factors were: type of housing and use of a feeder cup.

Table 5.25: Sensitivity and specificity values for $d_1$mft ≥ 3 at age 4-years for the CHAID analysis prediction tree (age 1-year) (n = 784).

$d_1$mft ≥ 3	
Sensitivity	69%
Specificity	60%

Figure 5.5: Diagrammatic CHAID risk assessment model for prediction at age 1-year of $d_1mft \geq 3$ at age 4-years (Se = 69%, Sp = 60%) (n = 784).



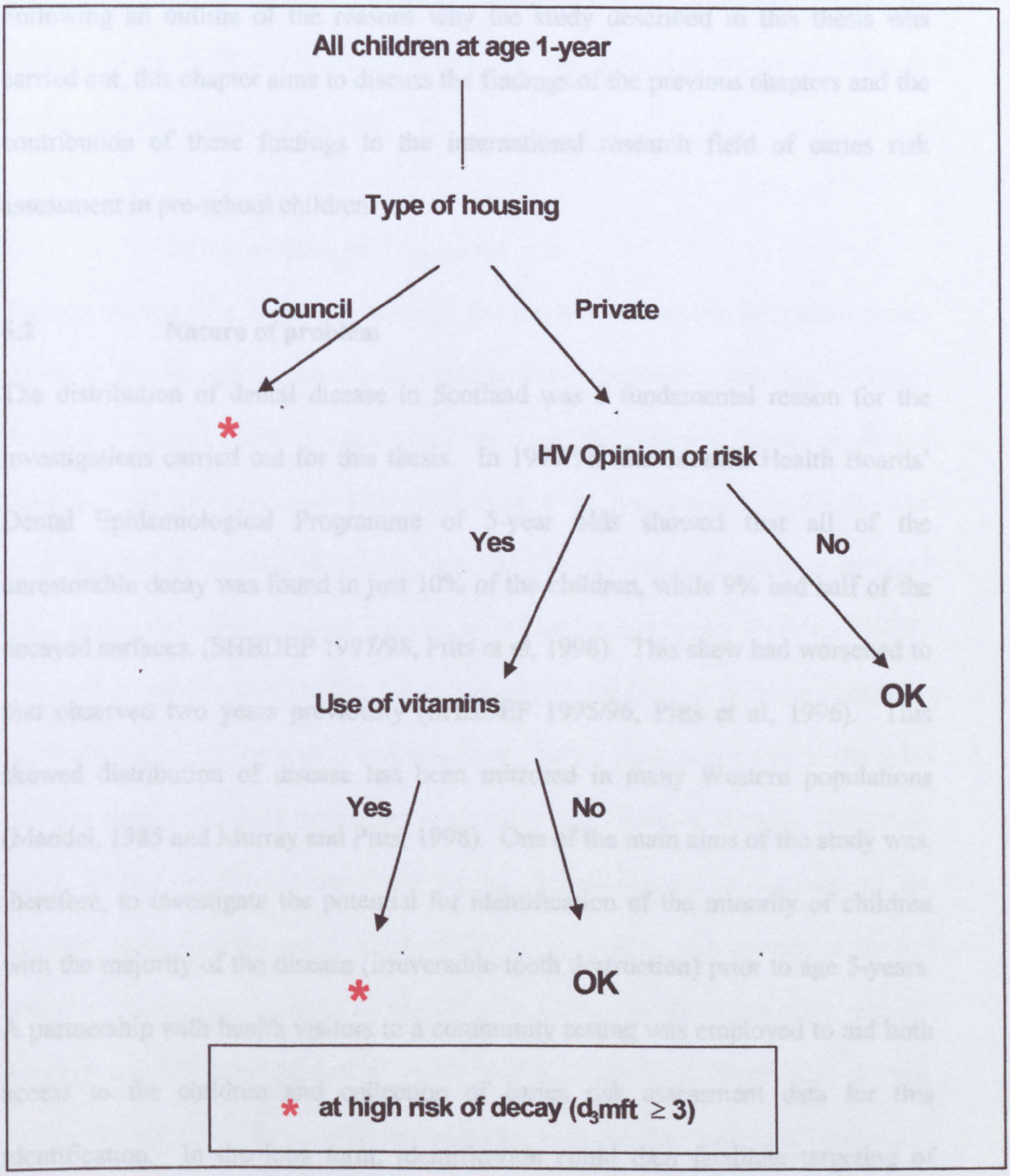
5.4.3.1.4 d_3mft at age 4-years ≥ 3 – 'high caries risk' model

The diagrammatic tree diagram of this risk assessment is shown in figure 5.6 and the sensitivity and specificity values in Table 5.26. The most predictive factors were: type of housing, HV opinion of risk and use of vitamins.

Table 5.26: Sensitivity and specificity values for $d_3mft \geq 3$ at age 4-years for the CHAID analysis prediction tree (age 1-year) (n = 784).

$d_3mft \geq 3$	
Sensitivity	65%
Specificity	69%

Figure 5.6: Diagrammatic CHAID risk assessment model for prediction at age 1-year of $d_3mft \geq 3$ at age 4-years (Se = 65%, Sp = 69%) (n = 784).



Chapter 6: Discussion

6.1 Introduction

Following an outline of the reasons why the study described in this thesis was carried out, this chapter aims to discuss the findings of the previous chapters and the contribution of these findings to the international research field of caries risk assessment in pre-school children.

6.2 Nature of problem

The distribution of dental disease in Scotland was a fundamental reason for the investigations carried out for this thesis. In 1997/98, the Scottish Health Boards' Dental Epidemiological Programme of 5-year olds showed that all of the unrestorable decay was found in just 10% of the children, while 9% had half of the decayed surfaces. (SHBDEP 1997/98, Pitts et al, 1998). This skew had worsened to that observed two years previously (SHBDEP 1995/96, Pitts et al, 1996). This skewed distribution of disease has been mirrored in many Western populations (Mandel, 1985 and Murray and Pitts, 1998). One of the main aims of the study was, therefore, to investigate the potential for identification of the minority of children with the majority of the disease (irreversible tooth destruction) prior to age 5-years. A partnership with health visitors in a community setting was employed to aid both access to the children and collection of caries risk assessment data for this identification. In the long term, identification could then facilitate targeting of preventive measures at those pre-school children with the greatest need. This has

been termed the application of a high risk strategy, as opposed to a whole population based strategy, where all individuals would receive a similar preventive regime, for example, water fluoridation (Hausen, 1994). A population-based strategy would be applicable where the distribution of decay followed a normal distribution pattern and the distribution of disease was not skewed. In many scenarios these two strategies can be carefully pursued in parallel.

6.3 Public health and deprivation

The application of a high risk strategy has many implications for public health services. The current literature shows an association between deprivation and both dental and general health (Carstairs and Morris, 1991; Sweeney, 1996; Hinds and Gregory, 1995, Gregory et al, 1995; Pitts et al, 1998; see also chapter 2.2.7). The most deprived individuals in society have the poorest record of health in terms of oral disease and other diseases, such as heart disease and stroke (Petersen, 1998). As noted previously (see chapter 1.1.3), there has been little or no improvement in the dental health of 5-year-olds in recent years, with a consistent group in the more deprived strata of society continuing to develop high levels of disease. If services could be directed toward these most needy individuals, the cost-benefit to the National Health Service as a whole might be enormous. At present, a general anaesthetic session in hospital for the removal of carious deciduous teeth requires the presence of a large number of highly trained medical and dental staff at great cost to the taxpayer. In general, dental caries in deciduous teeth is a preventable condition. If targeted preventive care following identification of caries risk in pre-

school children could reduce general anaesthetic lists by only one per week in an average general hospital, it is conceivable that dental costs might be reduced and the resources could be employed elsewhere in the Dental or wider Health Services. The long term result of targeted prevention could be a reduction in the caries prevalence of 5-year olds in Scotland, more effective use of health care resources, and achievement of the goals set out in the Oral Health Strategy for Scotland (The Scottish Office, 1995) and restated in a recent white paper (The Scottish Office, 1999). It must be emphasised that detailed cost-benefit analysis would be required to determine the validity of such speculations. However, for this type of effect to work in practice preventive based care must be available for these children. Recently, it has been suggested that the current payment system for NHS children's dentistry has failed to promote effective prevention amongst the most caries prone (Reekie, 1999). A system could, therefore, be developed to allow successful targeted prevention in order to reduce caries levels in 5-year-olds. It has been shown that dental health-related behaviours that either protect or put the child at risk of poor oral health are established in the pre-school years (Jones et al, 1996). Therefore, such a system must be employed at a very early stage, ideally at, or before, birth. Such a system would necessitate involvement of those health care personnel closely involved with children at very young ages or with expectant mothers.

6.4 A partnership with health visitors

Chapter 3 of this thesis explored the feasibility of a partnership with health visitors to achieve identification of those individual pre-school children at risk of developing dental caries. It was clear from the results (chapter 3.5) that health visitors were able to: 1) obtain consent for a large population-based longitudinal study of pre-school children; 2) achieve access to the children for collection of caries risk assessment data and 3) work in partnership with a study dentist to achieve this data collection. Previous studies have highlighted the ability of health visitors to both target and influence the dental health of young people (Bentley et al, 1992) as well as encourage early registration and dental attendance (Pine and Deas, 2000). Recent evidence has shown that 5-year-olds who were irregular attenders had significantly higher caries experience than those not registered, and that registration with a GDP does not equate to a 'healthy' attendance pattern (Tickle et al, 1999)). No studies, however, have focused on the feasibility of a partnership with health visitors for the caries risk assessment of pre-school children. Publication of the Nuffield report (Tyrell, 1993) revealed a need for the adoption of a multidisciplinary approach to the development of oral health professions and support personnel. Many authors have long recognised the role of health visitors in dental health (Simmonds, 1965; Blinkhorn, 1981; Stratford, 1979; Williams, 1980; Bentley, 1994 and Quinn and Freeman, 1994). In the government document, 'The Oral Health Strategy for Scotland' (The Scottish Office, 1995), a key proposal for reducing caries levels in 5-year olds was a "concentrated effort on pre-school children and their parents". The document highlighted the importance of health visitors within the community and

the possibility that they could gain access to this vulnerable age group. Blinkhorn (1981) recognised that the target group for dental health education programmes is the mother with a young child. Seward (1967) also emphasised the importance of education during the ante-natal period. This thesis has explored the ability of health visitors to achieve such access and has demonstrated that a partnership between dentists and such an important group of health services personnel, as part of the dental team, is indeed feasible.

Development of the risk models for this study (chapter 5.4.3) incorporated significant predictors (at age 1-year) of 'any' and 'high' caries risk in 4-year olds. One of these was the health visitor's (HV) opinion as to whether or not the child was at risk of developing caries. As described in chapter 4.3.3, this was a subjective assessment ('hunch') and the health visitors were given no formal training. They used their knowledge of the child's background, health and general living conditions. The fact that this opinion was so significant for three out of the four risk models, given that health visitors have little dental training, has far reaching implications for risk assessment of young children.

It was noted in 4.4.4 that the HV opinion of caries risk in year-2 of the study was required to complete the incomplete data sets for year-1 for this factor. This data supplementation was carried out as it was unlikely that opinion would have altered in the second year of the study and as noted in chapter 3.6.8.3, the health visitors' confidence in answering the question increased after year-1 of the study.

6.5 Methodology used for data collection

6.5.1 Dental examination

The dental examination technique used in this study was described in 4.1. As stated, examination did not involve the use of a dental mirror or probe. The teeth were not previously cleaned nor dried. One of the aims of this study was to assess the feasibility of obtaining access to large numbers of pre-school children in a community setting. The equipment used for dental examination was required to be minimal, transportable, quickly and easily assembled and packed and acceptable for use in a large number of homes and other environments. Essential for the technique of the dental examination was speed and reproducibility. The protocol described in the research proposal for the dental examination was, therefore, direct vision assisted by a pen-light and immediate recording of results, followed by data entry into a computer database. The reproducibility results (chapter 5.1.1.4) confirm that the technique used was reproducible.

6.5.1.1 Caries diagnosis

Caries diagnosis was carried out at the d_1 caries into enamel threshold to facilitate the assessment of enamel, dentinal and pulpal decay - this was described in chapter 4.1.1.1. As the results show, the use of the d_1 diagnostic criteria increased the level of detection of caries. Table 5.1 showed the caries prevalence for each of the four years of the study at the d_1 threshold of diagnosis (3%, 12%, 27% and 49% respectively) and, similarly, Table 5.2 at the d_3 threshold of diagnosis (0.4%, 4%, 12% and 33% respectively). Reproducibility of the caries diagnostic technique will

be discussed in section 6.6. Obviously, use of the $d_3$mft level of diagnosis underestimates the prevalence of decay, by as much as 16% at age 4-years, in this study population. If targeted preventive strategies were to be implemented on this 'underestimated' population they would, by definition, partially exclude some of the population with the greatest need for *primary* prevention – i.e. those with *reversible* carious lesions.

6.5.1.2 Access to the children for dental examination

The methods used to access the children for dental examination differed at years 1, 2, 3 and 4. These methods were fully discussed in 3.5.4. However, it is important to note that the use of these different access methods emphasised the true field nature of the study. The children examined were not only seen in a specific health centre but in various locations depending upon the age of the child and the family circumstances. The location of access was also dependent upon the health visitor, in terms of their home visiting arrangements and number of clinics organised within their individual health centres or doctors' surgeries. During the fourth year of the study, the majority of children were dentally examined in nursery school, therefore, the health visitor arranged a separate visit for saliva sampling and questionnaire completion. The study dentist, therefore, did not liaise with the health visitors in the fourth year to the same extent as in the earlier years of the study. However, the health visitors continued to independently collect caries risk assessment data without difficulty as evidenced by the similar amount of data was collected by the health visitors in the fourth year of the study compared to previous years (chapter 3.5).

This emphasised the ability of the health visitors to work in partnership with a dental health team to collect data without, however, the need for continual direction, but in the knowledge that support was available and provided when necessary.

6.5.2 Microbiological saliva sampling

A review of the literature on the methodological considerations of microbiological data collection was provided in 2.2.4.2. Krasse (1990) noted that the methodological problems associated with microbiological analysis should not be under-estimated. This part of the discussion outlines the reasoning behind the methodology used for microbiological data collection described in chapter 4.2.

In this study, microbiological sampling of the saliva was chosen rather than collection of plaque samples. Some authors have questioned the use of saliva (Van Houte 1993), others promoted its use (Krasse 1988). Krasse (1990) stated that the selection of the sampling method is dependent on the objective of the examination, age of the population and method used to cultivate the organisms. This study involved microbiological data collection of large numbers of 1, 2, 3 and 4-year old children in a community setting to facilitate analysis of its use in the caries risk assessment of this age group. The data was collected by health visitors during their daily practice and it was essential to find a technique of data collection which was not time consuming, relatively easy to perform and acceptable to the study children, the principle carer of the child and the child's health visitor. Saliva sampling rather than plaque sampling fulfilled these challenging objectives. The saliva sampling method of choice was the tongue-loop method (Beighton 1986) and was used

consistently for the duration of the study. Tanzer (1990) suggested that to achieve better microbiological predictive data, early carious lesions should be monitored. He also noted that cultivating samples by immersing semi-selective culture medium-coated supports in saliva can be problematic in terms of accuracy. Krasse (1990) emphasised the need for collaborative studies, as well as validation and control of the methods used. In order to confront many of the problems highlighted by these authors, the microbiological methodology for this study included a large sample size, caries detection at the d_1 threshold of diagnosis, use of traditional agar and confirmatory identification of micro-organisms at a distant established laboratory. This study did not include an investigation of basic salivary factors such as flow rate, buffer capacity and fluoride content. This was because available tests appeared inconclusive, too costly, complex and time consuming for application in a large community based caries risk assessment study. This was also a conclusion drawn from the UNCCRAS (Disney et al, 1992).

6.5.3 Study questionnaires

The methodological technique used to obtain the socio-demographic and health behaviour data was by means of questionnaires. Relating to dietary factors, Schou (1991) noted that the use of sugar consumption behaviour as a predictor of future caries presents problems, as methodological limitations and inadequacies impede the collection of valid and reliable data. The literature review on dietary factors (chapter 2.2.5) has shown that data collection methods differ. A number of researchers have used questionnaires (Ekman, 1990; Holbrook, 1993; Grindefjord et al, 1993; and

Kawabata et al 1997). Others have used the interview technique (Schroder and Granath, 1983; Schou and Uitenbroek, 1995; Roeters et al, 1995; and Ayhan 1996). Comparison with many of the studies described in the literature is, therefore, problematic. In relation to oral hygiene, Reisine and Douglass (1998) noted that a major problem confronting the investigation of the relationship between toothbrushing and ECC is the methodological issue of assessing the frequency of brushing, quality of plaque removal and actual levels of oral hygiene. Most reports of toothbrushing assess such questions by asking the primary caregiver. These reports are subject to recall bias, as well as to social desirability response bias. They suggested that the data on the relationship between toothbrushing and caries were equivocal and more attention should be directed at the development of more reliable and valid measures of oral hygiene, to more accurately assess the effect of this variable on caries risk. In the study described in this thesis the focus was placed on obtaining large amounts of data in a large, longitudinal, community based setting. Questionnaires were, therefore, the methodological tool chosen to collect this type of data. An important consideration was that the technique used by the health visitors to collect the data remained consistent for the 4-year study period.

The results described in 3.6.8 have shown the large numbers of completed parental and health visitor questionnaires returned for each year of the study. These high numbers returned indicate that the questionnaires were easily understood and completed by both parents and health visitors alike.

6.6 Reproducibility of data

6.6.1 Dental examination

Results of the reproducibility of the caries diagnostic techniques were provided in chapter 5.1.1.3. These included kappa values for both in vitro intra- and inter-examiner reproducibility and in vivo intra- and inter-examiner reproducibility. To summarise, the in-vitro intra-examiner reproducibility kappa values ranged from 0.62 – 1.0 (HBM) and 0.69 –0.81 (JP). In-vitro inter-examiner kappa values ranged from 0.37 – 0.71 (HBM v JP). Calibration in-vitro inter-examiner kappa values ranged from 0.92 – 1.0 (HB v CL) and 0.65 – 0.77 (JP v CL). In vivo intra-examiner kappa values ranged from 0.67 – 0.7 (HBM) and in vivo inter-examiner kappa values (HBM v JP) ranged from 0.67 – 0.70.

With one exception, the kappa values were all above 0.61. This represented 'substantial' examiner agreement (Landis and Koch, 1977). The one exception was a 'fair' value of 0.37 for in vitro inter-examiner reproducibility (HBM v JP) (see chapter 5.1.1.4.1.3, Table 5.8). This value, however, could be accepted as the kappa values for in vivo inter-examiner reproducibility (HBM v JP) were above 0.67. This showed that the examiners had acceptable agreement on examination of the actual study children. These ranges, coupled with the results from Table 5.5 that showed steady increase in vitro intra-examiner kappa values to 1.0 for the study dentist (HBM) over the duration of the study, support the reproducibility of the caries diagnosis data.

6.6.2 Microbiological saliva sampling

Results of the reproducibility of the microbiological methodology were provided in chapter 5.2.3. These showed that the study dentist (HBM) and study technician (VW) were able to identify caries associated microorganisms with excellent reproducibility value (all kappa scores 1.0, with one exception of 0.87) (Landis and Koch, 1977). Both intra-examiner and inter-examiner reproducibility scores were high. Confirmatory identification of microorganisms was carried out under the direction of Professor David Beighton in the laboratory of Kings College Medical School, London. These techniques were described in 4.2.6. The Standard Operating Procedure's (SOPS) for both culture and identification of caries associated microorganisms were assessed and affirmed by Professor Beighton (Appendix 4.15).

6.6.3 Study questionnaires

Section 5.3.2 presented the results of the questionnaire reproducibility. Kappa values for randomly repeated questions ranged from 0.46 to 1.0. The study questionnaires, therefore, had acceptable reproducibility.

6.7 Effect of doing things differently

The study methodology was fully described in chapters 3 and 4. As noted throughout this thesis, this study was a large scale (well over one thousand children), community-based, longitudinal study of very young children that involved working in partnership with a group of highly trained and respected health care professionals. Many of the methodological techniques were chosen in order to incorporate these

considerations. For example, the dental examination needed to be simple, quick, transportable and efficient. Different results may have been obtained if a more detailed examination could have been carried out in a clinic with increased lighting and added diagnostic equipment. However, it would not have been possible to involve such large numbers of children and many examiners would have been required, as was the case in other studies (Disney et, 1992; Grindefjord et al, 1995) or, alternatively, smaller numbers of children could have been used (Schroder and Granath, 1983; Schroder et al, 1994 and Reisine et al, 1994). Similar considerations were given to the saliva sampling technique. The tongue-loop method was chosen because of its simplicity, ease of use and acceptability to the health visitors. The results (chapter 5.4 and Appendices 5.3, 5.4 and 5.5) showed that the salivary caries associated microorganisms did not contribute sufficiently significantly to caries prediction to justify their future use in this capacity. However, more detailed plaque sample analysis might have increased their importance as risk factors. Salivary analysis such as buffer capacity, flow rate and mineral content was not carried out for this study and these more detailed techniques might also have been identified as significant caries predictors. It must be emphasised, however, that this study was community-based. It would not have been feasible to carry out every salivary and plaque test on a cohort of well over one thousand children for a period of four years. One of the main aims of the study was to explore a partnership with health visitors to collect risk assessment data and the methodological techniques had to be appropriate for use by health visitors within their already demanding daily case-load. The study questionnaires were also tailored to the health visitors. They never exceeded one

side of A4 paper and the questions were based on the health visitors' ability to access certain information on the children. The questionnaires were also developed with health visitor input (chapter 3.5). The possible combinations of questions which may yield significant risk markers of caries in pre-school children is enormous. It is possible, therefore, that other questions could have produced alternative, more sensitive and specific, risk models than those provided in chapter 5.4.

6.8 Risk model development and the implications for prevention of caries in pre-school children

One of the aims of this thesis was to develop a caries risk model for the identification of high risk pre-school children (4-year olds) in Scotland using a novel statistical approach. Krasse (1990) noted that any predictive test must possess at least three characteristics: validity, reliability and feasibility. In terms of validity, the model would ideally be both highly sensitive and specific to allow accurate prediction. Previously, in the literature, the specified ideal levels of sensitivity and specificity were 80% (Wilson and Ashley, 1989, Kingman, 1990, Hausen, 1997). The results of the logistic regression analysis for this study showed varied values (chapter 5.4.2). For the 'any risk' models values were $d_1mft > 0$, Se = 54%, Sp = 70% and $d_3mft > 0$, Se = 26%, Sp = 89%. For the 'high risk' models, values were $d_1mft \geq 3$, Se = 16%, Sp = 95% and $d_3mft \geq 3$, Se = 0%, Sp = 100%. The results of the CHAID analysis (chapter 5.4.3) also showed varied values. For the 'any risk' models values were $d_1mft > 0$, Se = 67%, Sp = 57% and $d_3mft > 0$, Se = 53%, Sp =

77%. For the 'high risk' models, values were $d_1$mft ≥ 3, Se = 69%, Sp = 60% and $d_3$mft ≥ 3, Se = 65%, Sp = 69%. As can be seen, all models have sensitivity and specificity values less than the suggested target minimum of 80%. The models produced by the CHAID analysis, compared with logistic regression analysis, had higher sensitivity values and less discrepancy between the sensitivity and specificity values. The 'high 'risk' models produced by logistic regression analysis had sensitivity values of only 16% and 0% for $d_1$mft and $d_3$mft models respectively compared with much higher values for the CHAID analysis (69% and 65% respectively) thus permitting a greater choice of 'high risk' models when using CHAID analysis. The risk models produced by the CHAID analysis for this study were, therefore, in terms of their predictive capability to identify caries risk pre-school children, the models proposed for use in a community setting.

The second characteristic that a predictive test must possess is reliability. This is synonymous with reproducibility. When the test is applied to the same subjects on different occasions, there should be a high correlation between the two sets of results. In terms of reproducibility of data collection, this was described and discussed previously (chapters 5.1.1.3. 5.2.3, 5.3.2 and 6.6). However, the importance of reliability of the risk model cannot be underestimated. The models developed must be tested on another population to ensure similar results could be obtained.

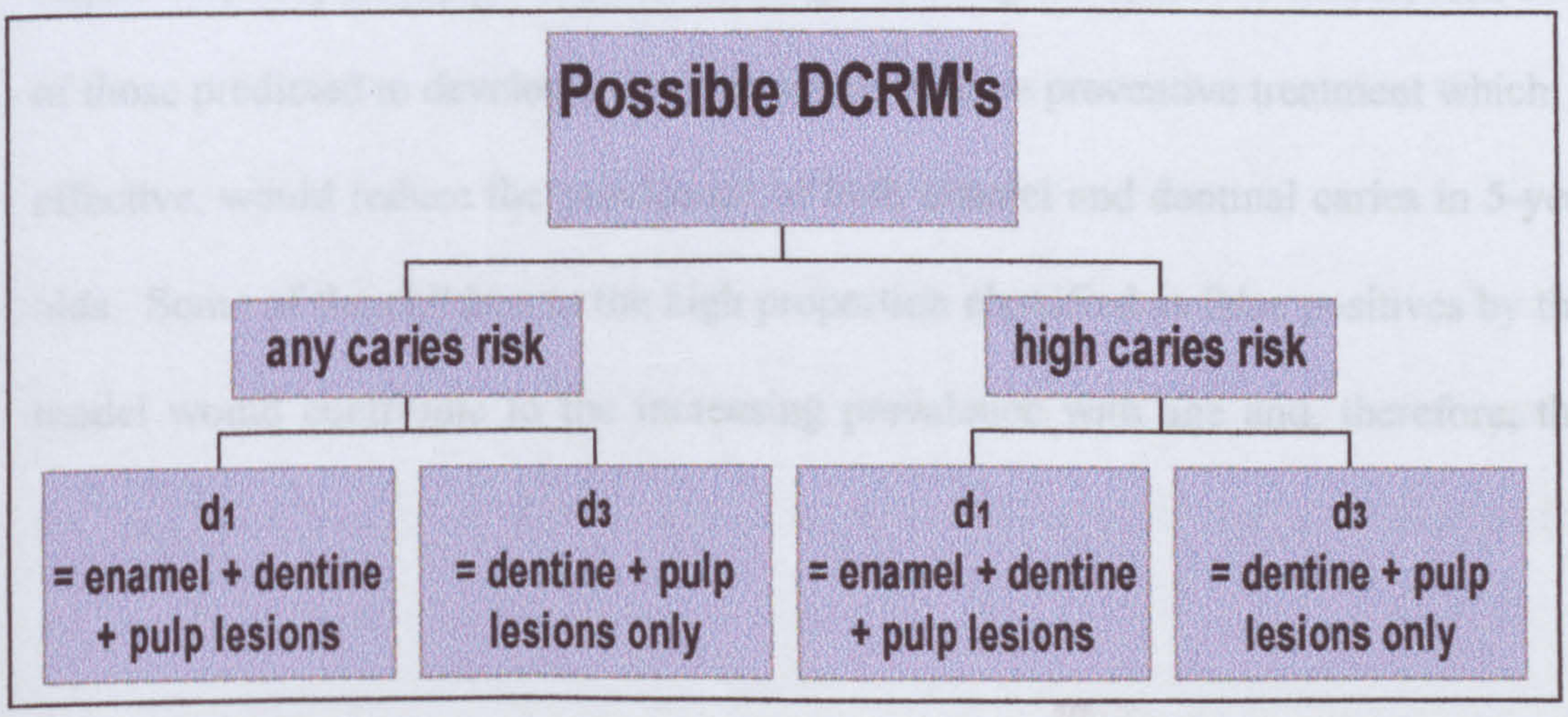
The third characteristic is feasibility, i.e. it should be inexpensive and easy to use. The final models developed from this study were based on a few simple questions. These questions can be easily administered in a community based setting by any

health care personnel. The caries associated microorganisms were not found to be significant enough predictors for use in the final models. This dramatically reduces the potential cost of the risk assessment and increases its simplicity for use in a community setting. The use of CHAID analysis requires a minimal amount of data in comparison with logistic regression analysis that needs the whole data set. Also, in a field setting a mathematical formula (usually needing a calculator or something more powerful for manipulation) is required to identify a child as 'at risk' using logistic regression analysis. Thus, in terms of ease of use in the community, a risk model derived from CHAID analysis would be a preferred model of choice for health visitors or any other health services personnel.

6.8.1 Discussion of the risk models developed by CHAID analysis

Four possible risk models were developed by the CHAID analysis in the development of a Dundee Caries Risk Model (DCRM). These have been presented in Figure 6.1.

Figure 6.1: Outline of development of possible Dundee Caries Risk Models (DCRM).



6.8.1.1 'any risk' model at the d_1 level of caries diagnosis ($d_1mft > 0$)

This model was presented in 5.4.3.1.1 (Figure 5.3). The model has a reasonable sensitivity value (67%) but a poorer specificity value (57%). It can, therefore, identify those children at risk of developing caries with reasonable accuracy (true positives). The poor specificity, however, means that a high proportion of false positives would be included in any targeted prevention. The d_1 caries prevalence at age 4-years within this population was 49%. Hausen (1997) stated that if the proportion of caries risk individuals in a population is close to half or more, this clearly implies that the occurrence of caries is not low enough to justify the effort and expense of identifying individuals at risk, although he refers to the prevalence at the d_3 level of caries diagnostic threshold. In such as situation the preventive efforts should rather be targeted at the whole population. Although this would appear to be sensible in practical terms, no cost-benefit analysis has been carried out in support of this statement. In Scotland, surveys show a d_3 caries prevalence of 57% for 5-year olds (Pitts et al, 1998) and 71% for 8-year olds (O'Brien, 1994). Therefore, in terms of the present study population, those 4-year olds with any caries (d_1) (prevalence 49%) have carious teeth likely progress to irreversible dentinal lesions (d_3) (see Figure 6.2). If primary prevention was targeted using the $d_1mft > 0$ model, then 67% of those predicted to develop d_1 lesions would receive preventive treatment which, if effective, would reduce the prevalence of both enamel and dentinal caries in 5-year olds. Some of the children in the high proportion classified as false positives by this model would contribute to the increasing prevalence with age and, therefore, this

prevention, if effective, would not be wasted expenditure, since some 'later' caries would be prevented.

A combined 'twin-track' approach using both population and high risk strategies would, therefore, be of benefit as even if the use of fluoride supplementation and toothbrushing became widespread there would remain a need for a high risk strategy, since a minority of the population would continue to develop a high proportion of dental disease.

The HV opinion of risk was one of the most predictive factors in this model which again emphasises the importance of the HV knowledge of the family. The DEPCAT score and parental smoking represented the second branch of the tree. Both of these factors represent social aspects of the family information which can be readily obtained. The predictive factors comprising the third branch of the analysis was breast-feeding and use of a dummy. The child was less likely to develop decay if breast-fed and did not use a dummy. The positiveve association between caries and the use of a dummy may be interpreted as the use of sweetened pacifiers.

6.8.1.2 'any risk' model at the d_3 level of caries diagnosis ($d_3mft > 0$)

This model was presented in chapter 5.4.2.1.2 (Figure 5.4). The model has a sensitivity of 53% and specificity of 77%. It has a satisfactory ability to identify those children who will not develop decay (true negatives) but has a poor ability to identify those who will develop decay (true positives). This model, therefore, includes a high proportion of false negatives (those predicted to be caries free who actually develop the disease). Targeted prevention using this model would not direct

prevention at these false negative individuals i.e. it would 'miss' nearly half of those for whom it could be of benefit.

The factors predictive of decay in this model include HV opinion of risk and parental smoking, which have been discussed previously. Food or drinks at night comprised the third branch of the tree. The child was at risk if this factor was positive. It would seem likely that this could be due to the presence of sugar in these food/drink intakes allowing the cariogenic bacteria the chance to produce acid at a time of low saliva production.

6.8.1.3 'high risk' model at the d_1 level of caries diagnosis ($d_1mft \geq 3$)

This model was presented in chapter 5.4.3.1.2 (Figure 5.5). It can be seen from the results that a reasonably satisfactory sensitivity value (69%) has been obtained at the cost of a poorer specificity value (60%). The reasonably high sensitivity value ensures that a reasonably high proportion of the children who actually develop the disease will be accurately identified and subsequently correctly targeted with caries preventive measures. However, the low specificity value means only 60% of the true negative children would be correctly identified and placed into the low caries risk group, resulting in a higher than ideal proportion of false positives, that is, those classified as high risk who are actually 'caries free', or with a caries level below 3 at 4-years. This would mean that these individuals would be included in the high risk group and receive preventive measures to apparently little or no purpose (Hausen, 1997). However, this high risk model was developed to identify those 4-year olds at 'high risk' of developing caries, that is 4-year olds with a $d_1mft \geq 3$. Those children

with a $d_1$mft of > 0 at 4-years of age will contribute to the overall caries prevalence of this age group (since there is 49% caries prevalence in the population at 4-years). Therefore, targeting of preventive measures to the group identified as 'high risk', even with a relatively low specificity – i.e. including a proportion at 'low risk' – would result in reduced caries in those at 'low risk' who contribute a not insignificant amount of caries to the overall prevalence. Hence, again, this prevention would not be 'wasted'.

The predictive factors included in this model were type of housing and use of a feeder cup. This was the only risk model developed which did not include the HV opinion of risk. The type of housing formed the first branch of the tree with those children in council housing at risk. If the child used a feeder cup at age 1-year, they were identified as 'low risk'. Type of housing can be indicative of social status and the use of a feeder cup allows drinks to be swallowed more rapidly than when a bottle is used. Identification of these factors can also be readily carried out in a community setting. One advantage of the absence of HV opinion of risk is that the data for this model could be collected in any environment, e.g. a dental surgery.

6.8.2.4 'high risk' model at the d_3 level of caries diagnosis ($d_3$mft ≥ 3)

This model was presented in chapter 5.4.3.1.3 (Figure 5.6). It has a sensitivity of 65% and a specificity of 69%. This model, therefore, has a reasonable capability of identifying both high caries risk (true positives) and caries free individuals (true negatives). This model was developed at the d_3 level of caries diagnosis in a population with a d_3 caries prevalence in 4-year olds of 33%. The target set by the

Scottish Office of 60% caries free 5-year olds was also for dentinal caries (d_3). The most recent survey of 5-year olds showed a d_3 caries prevalence of 57% (that is 43% free of dentinal decay - the value for Tayside was 42.7% caries free at d_3) (Pitts et al, 1998). It would appear, therefore, that a significant rise in decay occurs between 4- and 5-years of age. The results of this study showed an increase in d_3 caries prevalence from 0.4% at age 1-year to 4% at 2-years to 12% at age 3-years and then to 33% at age 4-years. Using this risk model, therefore, could potentially lead to a reduction of caries at age 4-years at the d_3 level of diagnosis. In addition this might also prevent d_1 (potentially reversible) lesions progressing to irreversible destruction. This would lead to a decrease not only in dentinal decay but also in the d_1 prevalence of disease in 5-year olds and beyond.

The significant predictors of decay in this model include type of housing and HV opinion of risk, which have been previously discussed. The use of vitamins was the factor forming the third branch of the tree. The use of vitamins was associated with high risk in this model. One possible explanation of this could be the sugar content of some vitamin syrups – the children were aged only 1-year and, therefore, the vitamins would almost certainly have been administered in liquid form. Another possible explanation is that of social status. It may be that those children who have been prescribed vitamins are in lower socio-economic groups and may, therefore, be compromised in terms of nourishment. The numbers involved in this branch of the tree were small – 14 in the high risk branch and 46 in the low risk branch - and the effect on sensitivity and specificity was to reduce sensitivity by 5% and increase specificity by 6%.

6.8.2 Development of the Dundee Caries Risk Model (DCRM)

One of the aims of this study was to develop a novel model for risk assessment of 4-year old pre-school children which could be used in a community setting to allow targeted preventive care (prior to irreversible tooth destruction) to those children at high risk of developing dental decay (chapter 2.3). The four individual models developed have been discussed in 6.8.2.1 – 6.8.2.4. By definition, and for the purposes of this thesis, the 'any risk' models would, therefore, not be proposed as the Dundee Caries Risk Model (DCRM). This study also aimed to develop a model which could allow targeted preventive care prior to irreversible destruction, i.e. primary prevention of decay. This requires the use of a d_1 threshold of caries diagnosis. The distribution of the decay within the study population (Table 5.3 and Figure 5.1) at a $d_1mft \geq 3$ was such that 27% of the population had 82% of the disease. Using this risk model would, therefore, allow targeted prevention to the minority of 4-year olds with the majority of the disease. The risk model for $d_1mft \geq 3$ at age 4-years has a sensitivity of 69% and a specificity of 60%. As discussed, a proportion of false positives would receive targeted prevention but these individuals would not necessarily be caries free. The d_1 prevalence of decay within the population was 49% (d_3 = 33%) but without a complete cost-benefit analysis it cannot be argued that targeting these high risk individuals is not practicable (Hausen, 1997). Applying this risk model in community setting requires answers to only two simple questions - type of housing and use of a feeder cup - and is, therefore, simple and easy to use in a community setting, without great cost to the

NHS. This risk model is reasonably valid, the data collected for its development reliable and is, finally, feasible in terms of cost and ease of use. These were the characteristics proposed for a risk model by Krasse (1990).

For the purposes of this thesis, therefore, the caries prediction model proposed as the DCRM is that for a $d_1mft \geq 3$ at 4-years of age (Figure 5.5).

6.9 Summary of literature review and general discussion

The literature review on caries risk assessment in relation to pre-school children is presented in chapter 2 of this thesis. It covered the use of individual factors and multiple factors in caries risk assessment. These factors can be grouped into dental, microbiological, socio-demographic, dental health behaviour and hunch factors. To summarise these individual factors, most authors agree that previous dental caries experience is one of the best indicators of future caries (Bader et al, 1986; Greenwell et al, 1990; Holbrook et al, 1993; O'Sullivan and Tinanoff, 1993; Reisine et al, 1994; O'Sullivan and Tinanoff, 1996; and Al-Shalan et al, 1997). Tinanoff (1995), however, noted that the presence of caries is a rather unsatisfactory risk assessment tool because it can only be used once a person is already affected by the disease. The unsatisfactory nature of this indicator is emphasised when the focus is on very young children, as is the case in this study. At 1-year of age there were few children with a dmf greater than zero (Table 5.1 shows that only 3% of 1-year olds had caries ($d_1mft > 0$), therefore, it was difficult to use this factor for the purpose of predicting future caries increment.

Microbiological factors have been consistently employed in caries risk assessment, with varying degrees of success. Some authors have found significant associations between specific microorganisms and both caries prevalence and increment (Bretz et al, 1992; Matee et al, 1992; Reisine and Litt, 1993; Thibodeau et al, 1993; Grindefjord et al, 1993; Kohler et al, 1995; Pienihakkinen and Jokela, 1995; Roeters et al, 1995; and Mattos-Graner et al, 1998). However, Isokanges et al (1993) stated that results from the literature suggest that microbiological tests contribute only marginally and are not cost-effective in the prediction of dental caries if clinical and socio-demographic data are available. Ansai et al (1994) also suggested that caries experience is difficult to predict by microbiological variables alone. Other authors have also questioned the value of salivary bacterial counts in risk assessment in pre-school children (Alaluusua and Renkonen, 1983; Schroder and Edwardsson, 1987; Holbrook et al, 1993; Schroder et al, 1994; and Lai et al 1997). Lai et al (1997) concluded that efforts to predict caries development in the primary dentition at an early age were not successful and a large field exists for research on caries prediction in young individuals. The results of the microbiological saliva sampling in terms of caries prediction of 4-year olds in this thesis did not find them sufficiently significant predictors to justify their use on a population - scale. They were, therefore, not incorporated into the final DCRM. It was important to collect microbiological data for these children as previous studies have shown their importance in young children (chapter 2.2.4). However, the results of this thesis would seem to mirror the view of Isokanges et al (1993) that microbiological tests

contribute only marginally and are not cost-effective in the prediction of caries if clinical and socio-demographic data are available.

Studies on caries risk assessment using dietary and oral hygiene factors in pre-school children are varied. Studies investigating the potential for sugar consumption in the prediction of caries have produced disappointing results (Persson et al, 1985; Schroder et al, 1994; Stamm et al, 1993; Holbrook, 1993; Reisine et al, 1994; and Grindefjord et al, 1995). It can be noted from the results in chapter 5.4 of this thesis that the food / drink consumption (at night) by the children at 1-year of age was a significant predictor of caries at 4-years of age. The type of food or drink consumed was not specified. It is probable, however, that since this was a significant predictor of caries, it was likely to be a sweet food or pudding and a milk or sugar-based drink. The Scottish diet is notoriously unhealthy (The Scottish Office, 1992) and much time and money has been invested in this area. The importance of dietary factors may also be masked within other factors, particularly social factors. It has been documented that poor infant feeding practice occurs to a greater extent in lower socio-economic groups (Silver, 1987) and immigrant status probably conceals unsuitable dietary habits (Grindefjord et al, 1995).

The literature on the use of oral hygiene in caries prediction appears somewhat inconclusive (Schroder and Granath, 1983; Paunio et al, 1993; Stecksen-Blicks and Holm, 1995; Ayhan, 1996; Febres et al, 1997; and Kawabata et al, 1997). In the study for this thesis none of the data collected on oral hygiene factors, such as toothbrushing and use of fluoride, were significant predictors of caries.

Although there is a wealth of information indicating that caries is concentrated in the lower socioeconomic status (SES) populations (Bailit, 1990, Tickle et al, 1999), few studies have analysed and reported the predictive value of social factors in children (Schou 1991). Recently, Reisine and Douglass (1998) concluded that data suggests increased risk of ECC in ethnic minorities, but few studies have addressed the joint effects of low SES or poverty status on ECC. Social factors were significant predictors of caries in this thesis. The postcode area in which the child lived was a significant predictor (DEPCAT), as was the type of housing (council rented or privately owned). Most of the social factors can be summarised as deprivation and this has been linked to higher caries in pre-school children in Great Britain (Hinds and Gregory, 1995, Moynihan and Holt, 1996, Sweeney, 1996). Postcodes have been recognised as useful markers of social class in Britain (Danesh et al, 1999). However, Ben-Shlomo and Smith (1999) and McLoone and Ellaway (1999) cautioned careful interpretation of Danesh et al's findings by stating that population characteristics should not be attributed to individuals and indicated that enumeration districts or postcode sectors should not be used as a proxy for an individual's social class.

An association between medical factors and increased caries risk has been shown by Holst et al (1997) and Peretz and Kafka (1997). However, other authors did not find such associations (Disney et al, 1992; Paunio et al, 1993; and Grindefjord 1995). None of the medical factors in this thesis were significant in a predictive capacity.

Subjective assessment or hunch factors have been investigated in some studies (Disney et al, 1992, Isokanges et al, 1993). However, no authors have investigated

the use of non-dental personnel to identify caries risk pre-school children using the hunch factor. As described in 6.4, the health visitor's hunch was one of the most significant predictors of high caries risk in 4-year olds.

The final section of the literature review looked at studies in which multiple factors were analysed to find the best combination of those individual factors that could be used to predict caries in children. This thesis aimed to develop a novel risk model for the prediction of caries in 4-year olds. To summarise, results from the literature have been disappointing. One possible reason for the poor correlations between various factors and caries increment prediction is the difficulties involved in caries diagnosis. The low sensitivity of visual techniques for approximal caries diagnosis in deciduous molars (Rimmer and Pitts, 1990 and Longbottom, 1992), which may represent a large proportion of caries, means that this disease might go largely unmeasured in pre-school children until late in the caries process. Schroder et al (1994) showed that with none of the predictors or combinations of predictors was it possible to find a screening level that combined high sensitivity with high specificity. Grindefjord et al (1995) achieved good results but, again, combined sensitivity and specificity of the logistic regression analysis was disappointing and the study showed that those at highest risk were those amongst the immigrant population. They concluded that immigrant background and sugar consumption should be considered as the most significant risk factors. This was in contrast to the UNCCRAS (Disney et al 1992), which found clinical variables to be the most significant – this was, however, in older children (see chapter 2.2.10). Grindefjord et al (1996) concluded that risk assessment in two steps before the age of 3.5-years

would be valuable in targeting children at high risk for early caries development but immigrant background and high sugar consumption should be considered as the most significant risk factors at age 1-year. By age 2-years, a test for mutans streptococci should improve the possibility to identify children most at risk. Stamm et al (1993) noted that, using logistic regression analysis, the models, both any risk and high risk, were below the current suggested minimum but for the high caries risk prediction, the sum of the sensitivities and specificities exceeded 1.40. Stewart and Stamm (1991) described preliminary risk assessment models developed by the UNCCRA study based on Classification and Regression Tree (CART) analysis and found the results encouraging. However, this study was carried out on older children. Reisine et al (1994) found that none of the demographic variables were significant but, en bloc, improved the ability of the discriminant analysis to predict caries at age 2-years from variables at age 1-year. Previous dmfs and *streptococcus mutans* levels were the most important predictors of future decay. In a previous study, Reisine and Litt (1993) found that race was a consistently important predictor and that *streptococcus mutans* was the most important predictor. The results provided in chapter 5 show that the most significant predictors of high caries risk (d_1 mft ≥ 3) in 4-year olds in Dundee-based data collected at 1-year of age, were: type of housing and lack of use of a feeder cup (DCRM) (for $d_3$mft ≥ 3, significant predictors were: type of housing; HV opinion of caries risk and the use of vitamins).

Although a vast number of related reports have appeared in the literature, there have been very few large-scale (n>200), multidisciplinary, population based, longitudinal

studies of caries risk assessment in pre-school children, especially in a demographic area similar to Scotland. Although the UNCCRAS was a unique and comprehensive study resulting in a set of prediction models, it was carried out on older cohorts of children and there has remained a need for a comprehensive caries risk assessment study of pre-school children which investigates both traditional and new methods of analysis. This thesis has provided an insight into the caries risk assessment of pre-school children in partnership with health visitors and the development of a novel risk model based on data collected in a community setting. There were differences between the risk models developed using logistic regression analysis and those using CHAID analysis in terms of their ability to predict high caries risk 4-year olds (chapter 5.4). The sensitivities (Se) and specificities (Sp) of the $d_1mft \geq 3$ caries risk model were 16% (Se) and 95% (Sp) (Logistic Regression) and 69% (Se) and 60% (Sp) (CHAID). The logistic regression analysis had a greater ability to predict those children who did not develop caries (true negatives) but only predicted 16% of those 4-year at high caries risk. The CHAID analysis, however, had a significantly greater ability to detect those 4-year olds at high risk of developing caries (true positives) and thus provides a greater future potential for the targeting of preventive measures to those children at highest risk of developing the disease. This result, in addition to the ease of use of CHAID in a primary care setting (since it requires no mathematical manipulation), promotes its use in this environment and for use by non-specialised health care personnel.

6.10 Prevention of caries in the pre-school child

The main purpose of developing a risk model would be to target preventive care to ward those with the greatest burden of disease. In a recent paper, Stecksen-Blicks and Borssen (1999) recommended that, depending on the availability of resources, groups or individuals who are high risk cases should be identified and provided with the intervention needed. Many different methods of caries prevention have been studied. It has been suggested that better dental health exists among children whose mothers had been given dental health education at home at an early stage of the child's life (Holt et al, 1985). Recently, Kowash et al (2000) concluded that regular home visits to mothers with infants to provide DHE to the mothers was shown to be effective in preventing the occurrence of caries in the infants. Twetman et al (1996) found that, although interpreted with caution, semi-annual fluoride varnish applications had a cariostatic effect in the primary dentition and might indicate that a fluoride varnish regimen is more cost-effective in areas with lower levels of fluoride in the drinking water. The need to develop methods to foster professional and public awareness of the importance of the weaning diet to dental health, especially in the high risk groups has been emphasised (Holt and Moynihan, 1996). Tickle et al (1999) concluded that regular attendance is conducive to good oral health and the results of their study support this view for 5-year-olds. They note, however, that the possibility exists that families displaying health visiting patterns may also exhibit other behaviours aimed at preventing dental disease. Various preventive strategies are thus available for targeted prevention and the effectiveness of some of these measures has been recently assessed in 12-year-olds (Hausen et al, 2000). This

paper concluded that "by offering all children only basic prevention, nearly the same preventive effect could have been obtained with substantially less effort and lower costs". They suggested that caution should be observed before implementing major shifts from the population strategy to the high-risk approach. However, the study was carried out on a much older cohort of children with an overall low caries prevalence and their use of microorganisms for selection of high risk groups is likely to have increased the cost of the risk assessment, although no cost-benefit analysis was reported.

6.11 Summary of findings of thesis

The findings of this study can be summarised as follows:

6.11.1 A partnership with health visitors

The results from chapter 3 have shown that it was feasible to work in partnership with health visitors to obtain access to pre-school children in Scotland for the purpose of collection of caries risk assessment data.

6.11.2 Risk model development

Chapter 5 provided both traditional and novel analysis of the data collected by the health visitors and study dentist to develop a caries risk model for high caries risk 4-year old pre-school children ($d_1mft \geq 3$) – the DCRM.

6.12 Interpretation of findings

6.12.1 A partnership with health visitors

The findings of this thesis (section 6.11) have important implications for the field of caries risk assessment in pre-school children. Firstly, they have shown that a partnership between dentists and other health care personnel is feasible. Not all countries have the type of health care professional described in this thesis, the health visitor. However, other forms of community based nursing are present. This study could not have taken place without such personnel. The large numbers of children recruited into the study (see chapter 3.6.3) can be attributed to the dedication of the health visitors, who were able to explain the study to the parents and obtain consent. In terms of the dental examination, it is probable that many of the families would not have allowed a study dentist to enter their home if not for the presence of the health visitor. The continued dedication of the health visitors to the study enabled them to sustain parental motivation and interest and much credit is due to them. This partnership with health visitors was a novel aspect of this thesis. The health visitors collected caries risk assessment data for a large number of pre-school children consistently over a 4-year period. If these health care professionals have the ability to collect such data, they could have the ability to carry out a caries risk assessment on a pre-school child and encourage the early initiation of targeted preventive measures toward the high risk children. This has far reaching implications in the research field of caries risk assessment. It may also be possible for health visitors to carry out a form of *clinical* caries risk assessment, as it has been documented in the

literature that personnel other than dentists can be trained to accurately examine young children briefly for decay (Lee et al, 1994).

6.12.2 Statistical analysis

A further novel aspect of this thesis which has an important contribution to caries risk assessment research was the statistical analysis for risk model development. The CHAID analysis has not been previously used to develop a caries risk model for pre-school children and the results from this study (chapter 5.7) show development of the Dundee Caries Risk Model (DCRM) with a sensitivity of 69% and a specificity of 60%. As previously discussed (section 6.8), these values, although slightly below the traditionally desired 80% (Kingman, 1992), have important implications for targeted preventive measures for Scottish pre-school children. Previous studies have focused on the use of logistic regression analysis (Stamm et al, 1992). The results of the logistic regression analysis for this study were shown in chapter 5.5. These indicated that CHAID analysis had a greater predictive capability than logistic regression analysis for this population.

6.12.3 Comparison with other prediction studies

In comparison with other caries prediction studies, the results of this thesis have a distinctive contribution. Dundee has no significant immigrant population – only 3.6% of the sample were from ethnic minorities. In Scotland as a whole the proportion of ethnic minorities is 1% (Office for National Statistics, 1996). The latter was previously shown to be one of the most significant risk factors

(Grindefjord et al, 1995) in a Swedish study on pre-school children. The study carried out for this thesis involved over one thousand pre-school children, followed longitudinally for 4-years, which no previous study has achieved. It was carried out in partnership with another team of health care professionals whose subjective assessment was one of the most significant predictors of caries risk in 4-year olds. Although other studies have analysed the clinician's ability to predict caries using his or her 'hunch' and a hygienist's hunch has also been explored (Disney et al, 1992), no research has involved health visitors, essentially non-dental personnel.

6.12.4 Application of results

The results of this thesis have implications in terms of application of these results within the community and in the field of prevention in the pre-school child. In the community, these findings may be used to develop a system of identification of high risk children as young as 1-year of age, which would be acceptable to both parents / guardians and to the front-line health care personnel carrying out the risk assessment procedure - in this country, health visitors. This would allow very young high risk children to be identified and directed into the dental health services system as early as possible, prior to the occurrence of irreversible tooth destruction. As Domoto et al (1994) noted, ignoring the presence of white spot lesions puts the preventively orientated researcher at a severe disadvantage. In terms of caries prevention, once the child is enrolled into the dental health care system, primary prevention (see chapter 1.2.1) would be the ultimate goal. However, once in the system both secondary and tertiary prevention could be carried out. The most important factor,

in terms of this thesis, is the early identification and initiation of preventive measures, a subsequent reduction in the caries prevalence of 5-year old children and an improvement in the dental health of Scottish children. However, as stated by Tinanoff (1995), risk assessment is a fundamentally different approach to patient care and successful implementation may require a significant effort, including school-wide consensus and quality assurance reviews, to assure implementation of the assessment and the subsequent preventive plan. This would also include more comprehensive training of health visitors on dental health in children, dental health education and prevention, as this does not seem to be currently achieved within the training system (Williams, 1980; Williams and Fairpo, 1982; Quinn and Freeman, 1994; Hunter et al, 1996; and Hunter and Chadwick, 1997). It has been reported, however, that local campaigns emphasising dental health in infants may increase how well informed health visitors are (Bentley, 1994) and increased cooperation between dental teams and health visitors has long been promoted (Seward and Goad, 1971).

6.12.5 Representativeness of data used for analysis

The numbers of children involved in this study were high (available cohort of 1890, 1683 consented in year-1, 1365 4-year olds dentally examined and 784 available child data sets available for production of DCRM). However, the children consented, dentally examined and child data sets used for risk model development should be representative of the available child population. The DEPCAT scores for the 11% of the available population *not* consented (n=207) showed a small but

significant difference compared with those children consented (n=1683), in that a higher proportion of those in lower DEPCAT categories (6 and 7) were not consented, compared to DEPCAT-1. This was not a surprising finding since families from areas of increased deprivation are recognised to be more difficult to access for health services purposes (Beal, 1990). However, the children dentally examined at age 4-years (n=1365) and the child data sets used for development of the DCRM were deemed to be representative of the available cohort (n=1890) (including those not consented) for the following reasons:

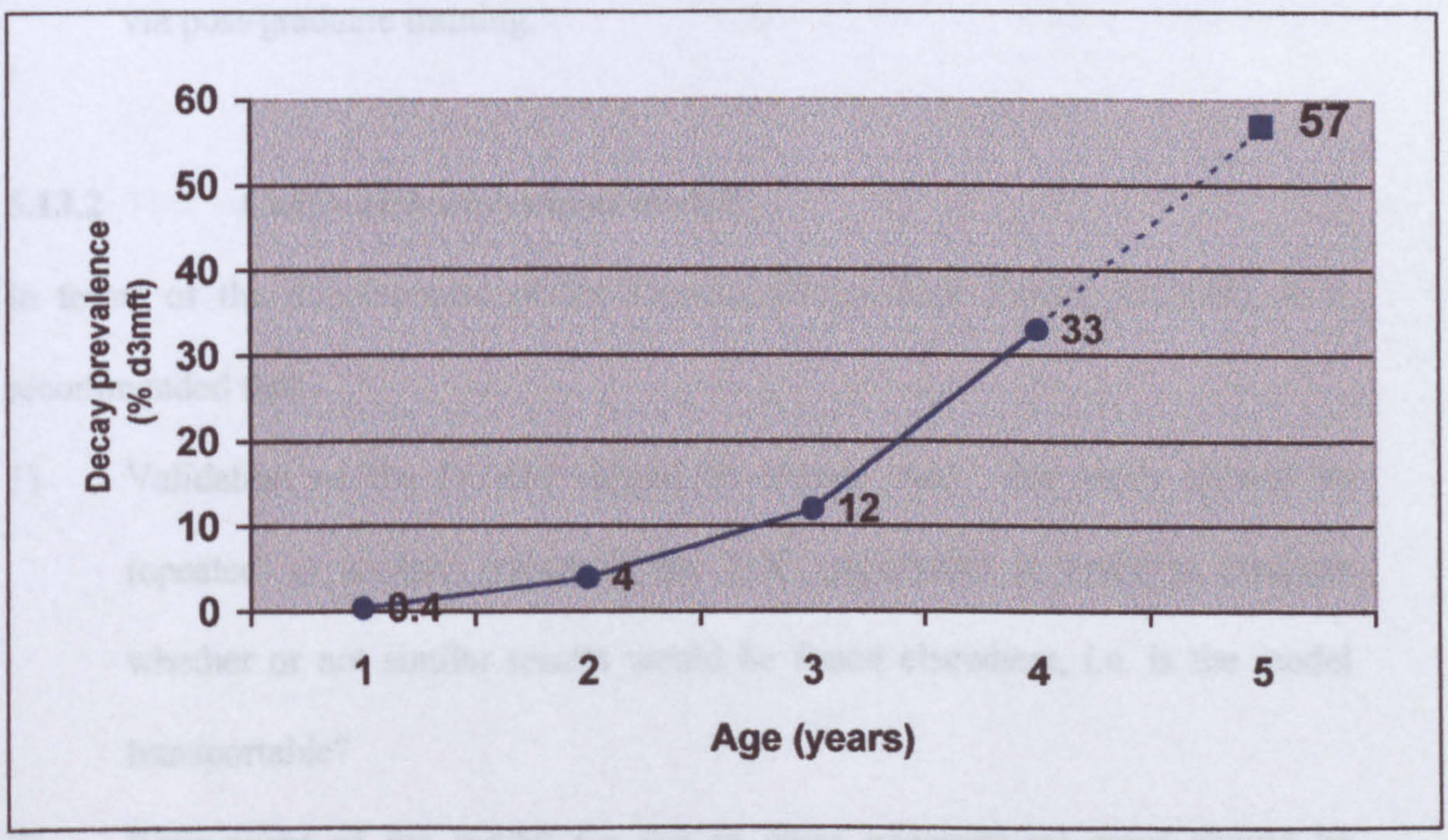
1) The prevalence of decay (d_3) in the study population at age 4-years (see Figure 6.2) follows a curve which continues linearly when extrapolated to the most recent SHBDEP figure for decay prevalence in 5-year olds (in Tayside) (Pitts et al, 1998). This suggests that, the caries diagnostic methodology used in this study is comparable to that used in the SHBDEP surveys and that the sample examined in this study is representative of the child population in this geographical area.

2) The DEPCAT values for those consented children dentally examined (n=1365) and those not dentally examined were not significantly different (see Appendix 5.6).

3) Of those children dentally examined and whose data was used to develop the DCRM (n=784), there was a DEPCAT skew, i.e. the higher the DEPCAT score (6 and 7) the less likely these children appeared in the DCRM (see Appendix 5.6). However, the mean $d_1$mft and $d_3$mft values at age 4-years were not significantly different for those children in, or out of, the DCRM

(see Appendix 5.6). Thus, the DEPCAT skew in terms of those in, or out of the DCRM, does not skew the caries prevalence data, i.e. the effect of the skew on d_1 and $d_3$mft was not significant.

Figure 6.2: Prevalence of dental caries at the d_3 threshold of diagnosis for the study cohort at ages 1, 2, 3 and 4-years and the SHBDEP figure for 5-year olds in Tayside (1997/98).



6.13 Recommendations and future research

6.13.1 Health visitors and dental health

In the future, it is recommended that health visitors (or similar health care personnel) have front-line involvement in relation to the dental health of pre-school children. This would involve the:

1) Ability to carry out the caries risk assessment of pre-school children

2) Appropriate direction of high risk children to oral health care services.

3) Training of health visitors in order that they could deliver a consistent and high standard of oral health promotion to all parents / guardians. This training would need to be carried out in colleges / universities and updated via post-graduate training.

6.13.2 Caries risk assessment model

In terms of the development of the Dundee Caries Risk Model (DCRM), it is recommended that:

1) Validation of the DCRM should be carried out. The study should be repeated on another cohort of the U.K. population in order to evaluate whether or not similar results would be found elsewhere, i.e. is the model transportable?

2) Refinement of the model for use in other geographical areas should be carried out. Other questions, for example, immigrant status and dental attendance, should be added which might lead to refinement of the model.

3) The DCRM should be tested. It is recommended that the model be used to identify high risk individuals in a population, followed by double-blind administration of preventive measures to evaluate the effectiveness of the preventive measures on the caries prevalence and incidence in comparison to those of a controlled sample of that population.

6.13.3 Caries risk assessment of pre-school children

The following recommendations are made for the field of caries risk assessment:

1) That full data exploration, e.g. investigation of novel analytical techniques and step-wise prediction analysis, be carried out on prospective longitudinal data for very young children (< 5-years) to develop highly sensitive and specific risk models applicable to a community based setting.

2) That non-dental personnel, such as health visitors, in close contact with young children be included in future studies.

Chapter 7: Hypotheses tested and conclusions

7.1 Introduction

This concluding chapter aims to answer the two research questions postulated in chapter 1.3 through the testing of several hypotheses and provide a list of the conclusions of the thesis.

7.2 Hypotheses for thesis tested

Hypothesis 1.1

*That it is **feasible** to employ existing health services personnel to access pre-school children in order to collect caries risk assessment data for a 4-year longitudinal caries risk assessment study in partnership with a study dentist.*

The results presented in chapter 3.5 showed that for a period of four years the health visitors consistently gained access to over one thousand pre-school children and their parents / guardians for consent, microbiological saliva sampling and questionnaire completion (in partnership with a study dentist carrying out dental examination of these children). No extra personnel were recruited to carry out these tasks and the health visitors fitted the tasks within their daily duties.

Thus the hypothesis is proved

Hypothesis 1.2

*That it is **feasible** to develop a multi-factorial caries risk prediction model in order to identify (to a reasonable degree of sensitivity and specificity) pre-school children at high risk of developing dental caries.*

The results provided in chapter 5, showed the development of a caries risk prediction model for 4-year old children (n = 784) at the d_1 threshold of caries diagnosis for those children with a dmft ≥ 3, the DCRM. This DCRM had a sensitivity of 69% and a specificity of 60%. These values, although not ideal, can be considered reasonably high against others in the literature.

Thus the hypothesis is proved.

Hypothesis 3.1

*That health visitors in Dundee can be **recruited** to participate in a 4-year longitudinal caries risk assessment study of pre-school children.*

All the health visitors in Dundee agreed to participate in the study and continued to collect data for the 4-year duration.

Thus the hypothesis is proved.

Hypothesis 3.2

*That the health visitors can gain **consent** for a 4-year longitudinal caries risk assessment study of pre-school children.*

Health visitors gained consent for 1532 from a total of 1890 children born and resident in Dundee between 1 April 1993 and 31 March 1994. This represented a consent rate of 89%. At the close of the study, the consent rate was still a remarkable 89%.

Thus the hypothesis is proved.

Hypothesis 3.3

*That it is **feasible** to employ existing health visitors to **collect** caries risk assessment data (involving microbiological saliva sampling and questionnaire completion) for the majority of a large cohort of pre-school children for a 4-year longitudinal caries risk assessment study.*

All the health visitors for Dundee obtained 1436, 1381, 1247 and 1150 saliva samples (85%, 82%, 74% and 68% of those consented), 1426, 1394, 1261 and 1163 health visitor questionnaires (85%, 83%, 75% and 69% of those consented) and 1405, 1342, 1250 and 1149 parental questionnaires (83%, 80%, 74% and 68% of those consented) for the children at ages 1-, 2-, 3- and 4-years of age, respectively.

In response to a feedback questionnaire (see chapter 3.5.9), the health visitors did not encounter any great difficulties with the collection of this data and were able to fit these tasks into their daily duties.

Thus the hypothesis is proved.

7.3 General research questions answered

Research question 1

Can a study dentist work in partnership with health visitors to gain access to a consented cohort of pre-school children for the purpose of caries risk assessment?

Answer: Yes. Hypotheses 1.1, 3.1, 3.2 and 3.3 are proved.

Research question 2

Can pre-school children at high caries risk be identified (through such a partnership) using dental, microbiological, dietary, oral hygiene, social, medical and hunch factors?

Answer: Yes. Hypothesis 1.2 is proved. However, the model has a sensitivity of 69% and a specificity of 60%.

7.4 Conclusions of thesis

7.4.1 Conclusion 1

A partnership between dentists and health visitors can be employed to access large numbers of pre-school children for the purposes of a prospective, longitudinal, caries risk assessment study.

7.4.2 Conclusion 2

A caries risk assessment model (DCRM) with a sensitivity of 69% and a specificity of 60% was developed which could enable high caries risk 4-year olds to be identified in a community setting.

7.4.3 Conclusion 3

Identification of high caries risk 4-year olds could allow targeted prevention toward this risk group prior to the initiation of irreversible dentinal caries and a reduction in the prevalence of caries in Scottish 5-year olds may, therefore, be attainable.

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Addresses of Manufacturers

Don Whitley Scientific Limited: 14 Otley Road, Shipley, West Yorkshire, England.

BD17 7SE. Tel: 01274595728

Camlab Limited: Nuffield Road, Cambridge. CBL 1TH.

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Jencons Scientific Limited: Cherrycourt Way Industrial Estate, Leighton Buzzard,

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Pro-Lab Diagnostics: 10a Croft Business Park, Bromborough, Mersyside, England.

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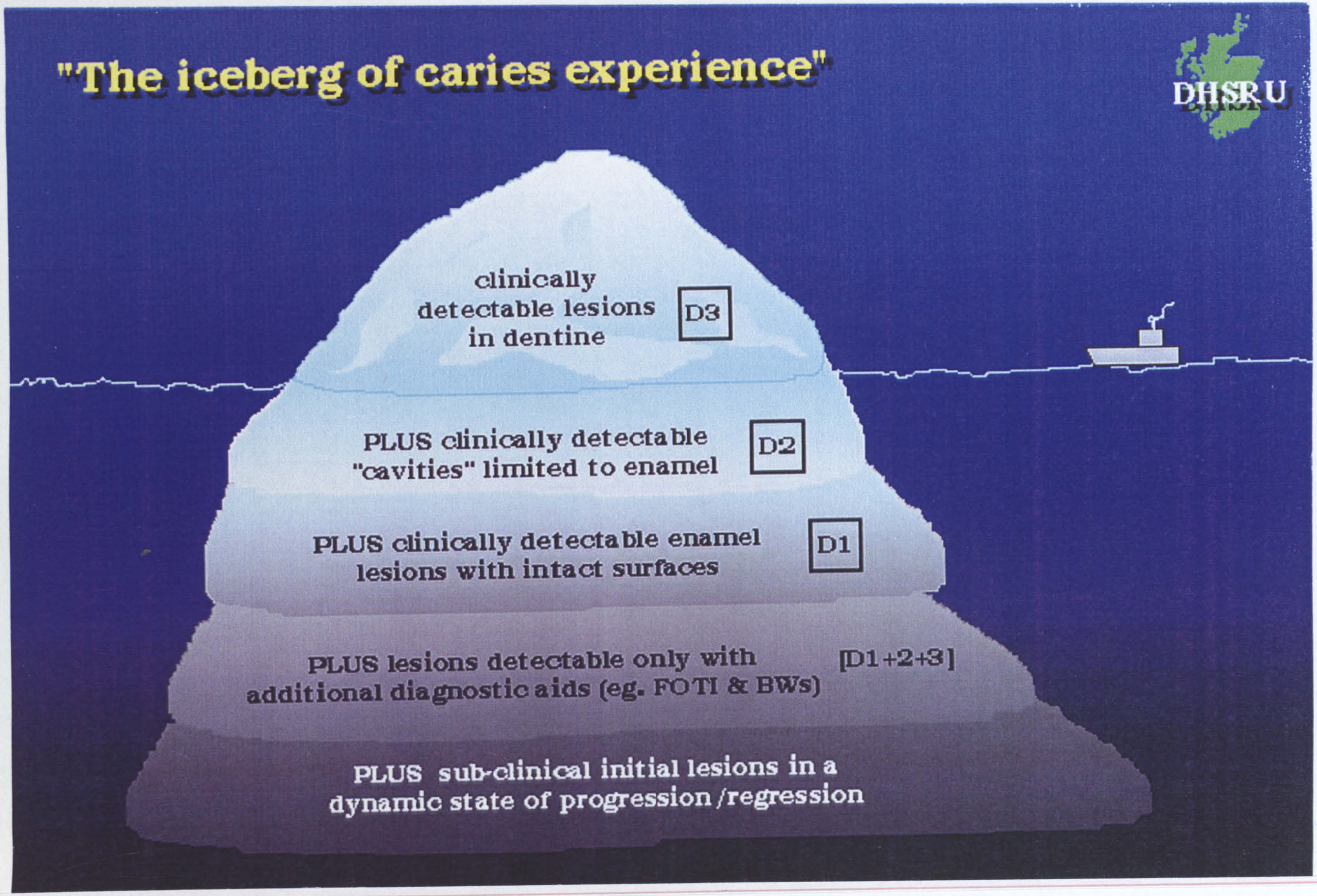
Appendices

1.1 Representation of different caries diagnostic thresholds in the form of an iceberg



Appendix 1.1 Representation of different thresholds in the form of an iceberg (reproduced with kind permission from Professor N B Pitts)

3.1 Tayside Health Board health visitor information leaflet

HEALTH VISITORS

WHAT OUR WORK INVOLVES.....

- ANTE-NATAL VISITING AND ADVICE
- CHILD GROWTH AND DEVELOPMENT MONITORING
- HOME VISITING SERVICE
- SPECIAL CLINICS e.g. SLEEP/OBESITY
- PARENTCRAFT CLASSES
- HEALTH PROBLEM COUNSELLING
- BEREAVEMENT VISITS
- ELDERLY VISITING
- HANDICAPPED VISITING
- ACCIDENT PREVENTION
- IMMUNISATION
- ETC...

Works with Other Agencies

IN HOSPITALS

Health Educators

IN HEALTH CENTRES

Works to Prevent Disease and Ill Health

IN HOMES

Fully Qualified Nurses

IN SCHOOLS

Members of your Primary Health Care Team

IN CLINICS

Appendix 3.1 Tayside Health Board health visitor information sheet

3.2 Consent letter issued at child's 8-month developmental screening

CONFIDENTIAL

- **Consent Form - University of Dundee Dental Hospital and School**

Dear Mother/Guardian

The University of Dundee Dental School is carrying out a study to find out if it is possible to predict, at a very early age, which children will develop tooth decay (dental caries) as they grow up. In order to do this we are hoping to examine, over a 4 year period, every child born and resident in the Dundee area between 1 April 1993 and 31 March 1994. At ages 1 year, 2 years, 3 years and 4 years each child will be examined by a Health Visitor and a Dentist. The Health Visitor will take a small sample of tongue saliva from each child's mouth, as well as looking at the number of the child's teeth present. In addition, the Health Visitor will complete a form providing confidential medical and social information. She will also ask one of the child's parents to complete a simple questionnaire. The Dentist, at this or a separate visit, will carry out a detailed examination of the child's teeth. All information gathered for the study will be treated as confidential and in accordance with the terms of the Data Protection Act.

Since your child was born during the chosen period we are studying, it would be most helpful if you agree to your child taking part in the study. You would, of course, be free to withdraw your child from the study at any time without giving a reason and without this effecting your child's future dental care. The results from this study will be used to develop local dental services for children.

Yours sincerely

Professor N B Pitts **Dr J Radford** **Dr C Longbottom** **Mrs M Robertson**

I.....mother/guardianof (child's name).....

(child's date of birth)..... (child's address).....

.....

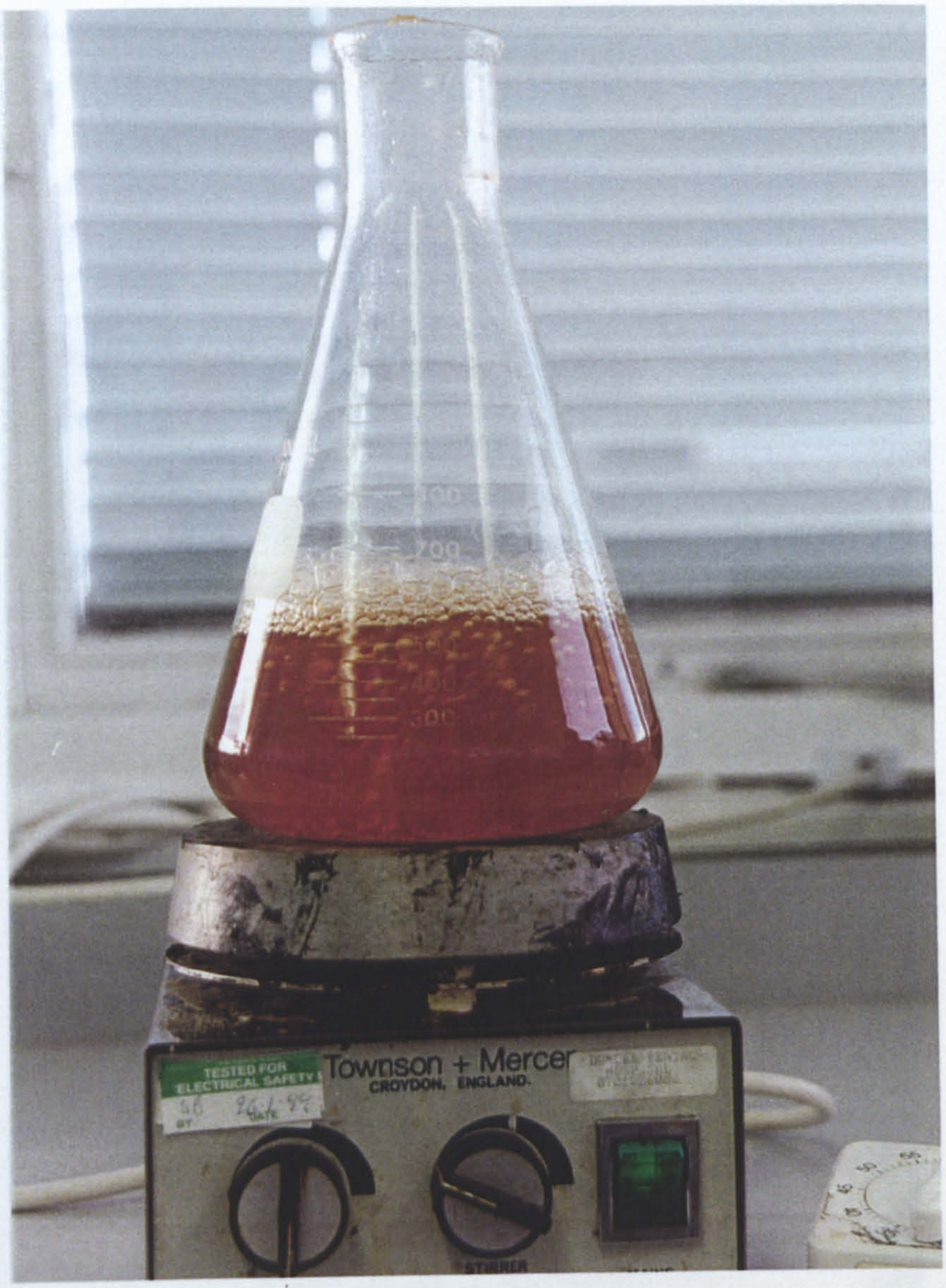
have read and understood the above and do/do not (delete as appropriate) consent to my child taking part in the study.

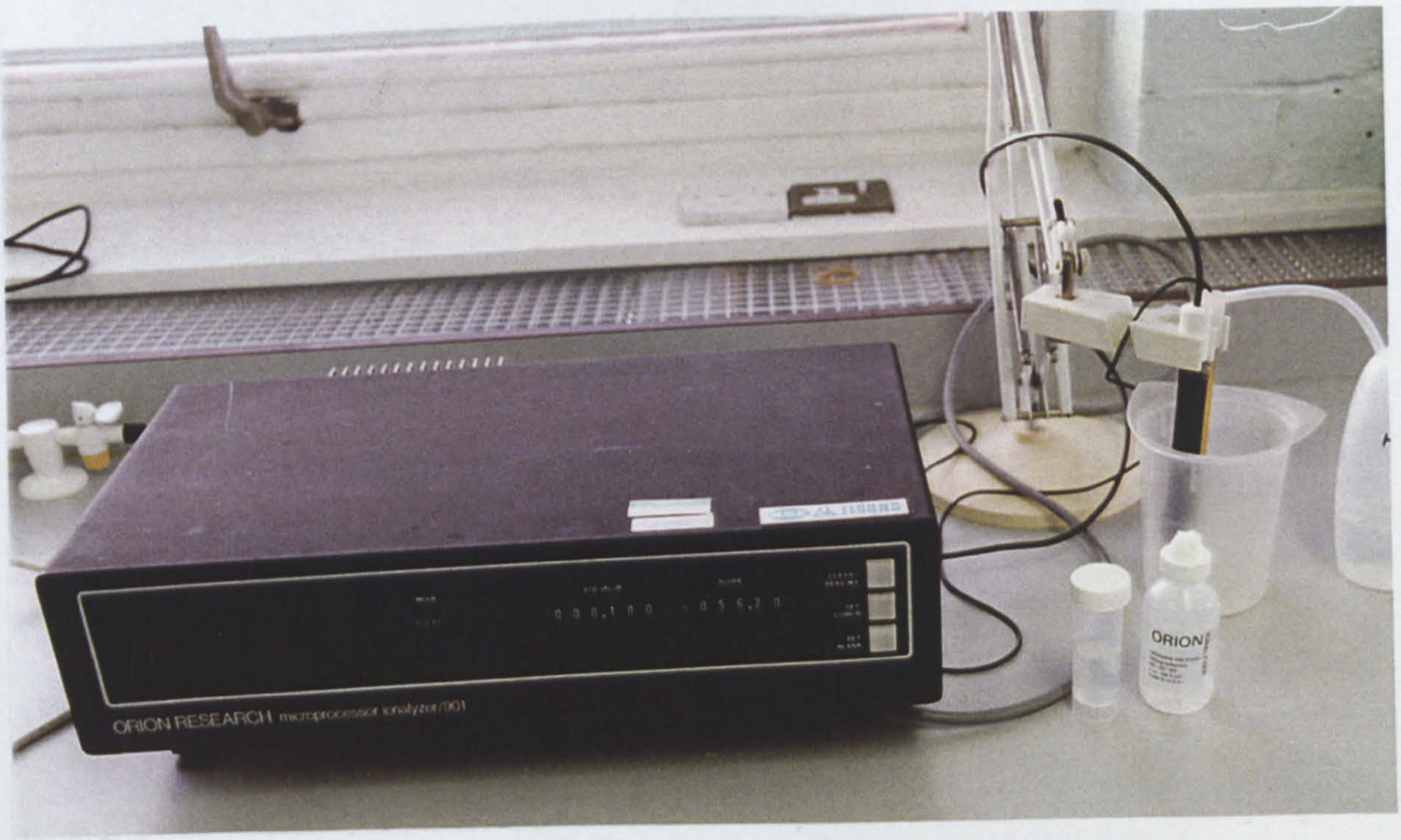
Signed ..... Date ...../...../.....

3.3 Photographs of laboratory equipment

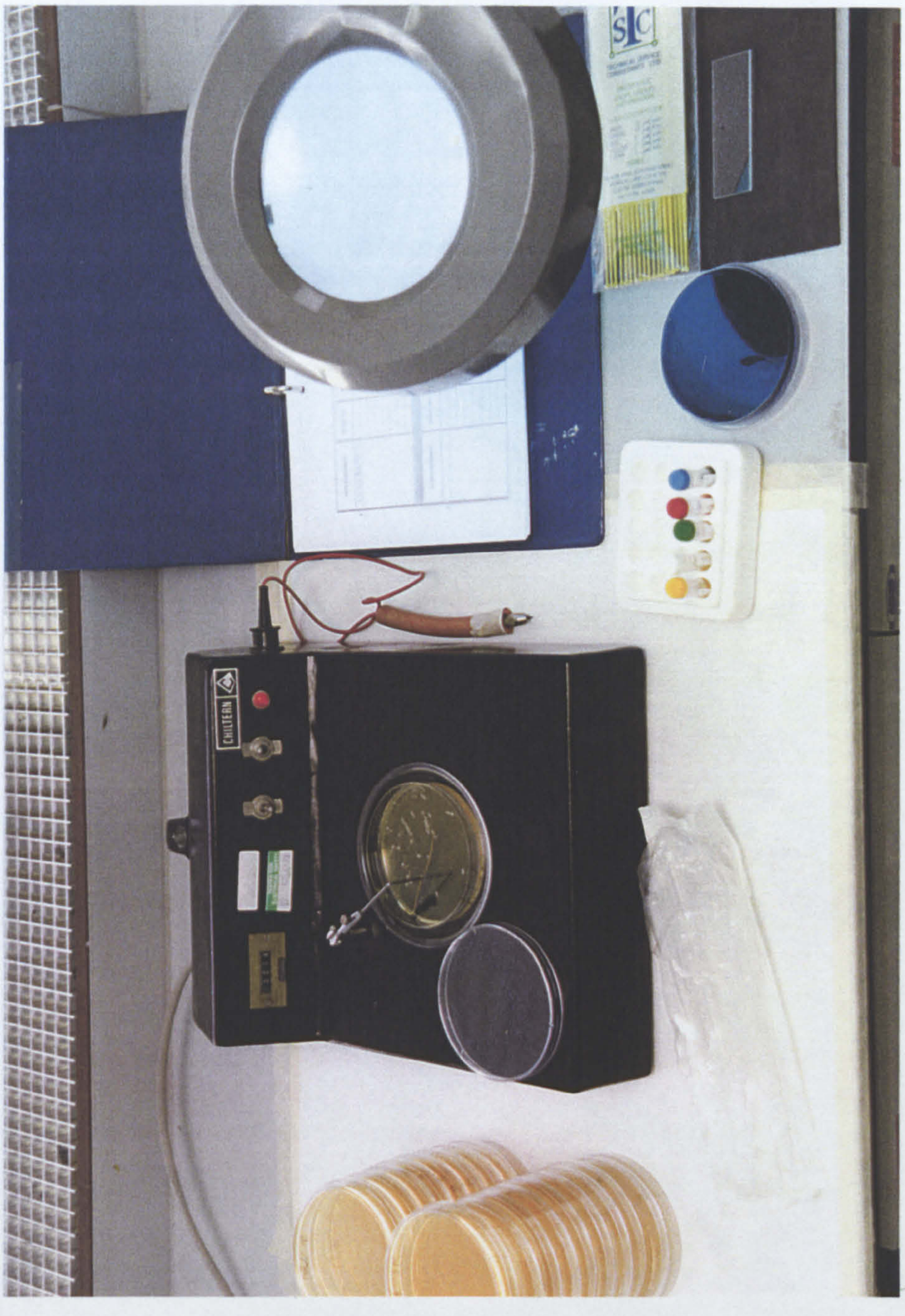
Appendix 3.3 Photographs of laboratory equipment

Appendix 3.3 Photographs of laboratory equipment





Appendix 3.3 Photographs of laboratory equipment



Appendix 3.3 Photographs of laboratory equipment

3.4 Photographs of health visitor training sessions

Appendix 3.4 Photographs of health visitor training sessions

Appendix 3.4 Photographs of health visitor training sessions

Appendix 3.4 Photographs of health visitor training sessions

3.5 Photograph of microbiological saliva sampling technique

Appendix 3.5 Photograph of microbiological saliva sampling technique

3.6 Parental questionnaires for each of the four years of the study

HEALTH VISITOR / MUTANS STUDY

Study No.

PARENTAL QUESTIONNAIRE (12 MONTHS)

CONFIDENTIAL

Thank you for your cooperation in this study. Please answer the following questions as accurately as possible and return the form to the Health Visitor. All information is completely confidential and for research purposes only.

To be completed by parent / guardian. Today's date:..... /...../.....

Q1 Was your child bottle-fed or breast-fed? (Please circle which one or both)

If yes, for how long? breast...../ bottle..... (to nearest month)

Q2 (i) Please circle the following meals your child has per day

breakfast dinner tea supper

(ii) Between meals, in general how many times per day does your child have a

sugar-containing (a) hot drink?..... (b) cold drink?.....

and in general, which ones .....

Please circle which of the following your child generally uses for drinking

bottle feeder cup cup

(iii) Does your child have a snack (e.g. a biscuit) with any of these drinks? Yes/No

If yes, on how many occasions per day?..... and please circle the snack(s) generally given:

sweet biscuits sweets crisps fruit other.(please specify) .....

(iv) Please circle which of these your child has at bedtime:

drink snack both none

(v) Which of these does your child have during the night:

drink snack both none

Q3 (a) Are your child's teeth brushed? Yes/No

(b)If yes, how often..... (c) And by whom? .....

(d)Is toothpaste used? Yes/No

(e)Do you give your child fluoride tablets/drops daily Yes/No

Q4 (i)Does your child attend day-care/nursery more than twice per week? Yes/No

If yes, please name facility .....

(ii)Is your child in the care of a relative/childminder more than two half-days / two evenings per week? Yes/No

Q5 When you carry out the bulk of your food shopping, **how** do you travel to the shops?
Please circle one of the following:

(a) on foot (b) by bus (c) by car (d) by taxi

Thank you for your help with this study

Study Number

HEALTH VISITOR / MUTANS STUDY

CONFIDENTIAL **PARENTAL QUESTIONNAIRE (2 YEARS)**

Thank you again for your cooperation in this study. Please answer the following questions as accurately as possible and return the form to the Health Visitor. All information is completely confidential and for research purposes only. To be completed by parent / guardian.

Today's date: ☐**dd** ☐**mm** ☐**yy**

Q1 Is your child still:

bottle fed yes☐ **no** ☐ **breast fed yes**☐ **no** ☐ **none? yes**☐

Q2 (i) Which meals has your child each day: **breakfast yes**☐ **no** ☐ **dinner yes**☐ **no** ☐

tea☐ **yes**☐ **no** ☐ **supper yes**☐ **no** ☐ **none yes**☐ **no** ☐

(ii) Between meals, in general how many times per day does your child have a:

hot drink ☐ **cold drink?** ☐

If so, which type(s).....

What does your child generally use for drinking:

bottle yes☐ **no** ☐ **feeder cup yes**☐ **no** ☐ **cup? yes**☐ **no** ☐

(iii) Does your child have a snack with any of these drinks? **yes**☐ **no** ☐

If yes, on how many occasions per day? ☐

Which snacks are given:

biscuits yes☐ **no** ☐ **sweets yes**☐ **no** ☐ **crisps yes**☐ **no** ☐ **fruit yes**☐ **no** ☐

other?.....(please specify)

(iv) Which of these does your child have at bedtime:

drink yes☐ **no** ☐ **snack yes**☐ **no** ☐ **none? yes**☐

(v) Which of these does your child have during the night:

drink yes☐ **no** ☐ **snack yes**☐ **no** ☐ **none? yes**☐

Q3 (a) Are your child's teeth brushed? **yes**☐ **no** ☐

(b) If yes, how many times per day? ☐

(c) By whom? **parent yes**☐ **no** ☐ **child yes**☐ **no** ☐ **other? yes**☐ **no** ☐

(d) Is toothpaste used? **yes**☐ **no** ☐

(e) Do you give your child:

fluoride tablets yes☐ **no** ☐ **fluoride drops daily yes**☐ **no** ☐ **none? yes**☐

Q4 (i) Does your child attend day-care/nursery more than twice per week: **yes**☐ **no** ☐

If yes, please name facility .....

(ii) Is your child in the care of a relative/childminder more than two half-days / evenings per week: **yes**☐ **no** ☐

Q5 When you carry out the bulk of your food shopping, how do you travel to the shops?

on foot☐**1** **by bus**☐**2** **by car**☐**3** **by taxi**☐**4**

THANK YOU for your help with this study

Study Number ☐☐☐☐☐☐

HEALTH VISITOR / MUTANS STUDY

CONFIDENTIAL **PARENTAL QUESTIONNAIRE (3 YEARS)**

Thank you again for your cooperation in this study. Please answer the following questions as accurately as possible and return the form to the Health Visitor. All information is completely confidential and for research purposes only. To be completed by parent / guardian.

Today's date: ☐☐**dd** ☐☐**mm** ☐☐**yy**

Q1 Is your child still:

bottle fed yes☐ **no** ☐ **breast fed yes**☐ **no** ☐

Q2 (i) Which meals has your child each day: **breakfast yes**☐ **no** ☐ **dinner yes**☐ **no** ☐

tea yes☐ **no** ☐ **supper yes**☐ **no** ☐

(ii) Between meals, in general, how many times per day does your child have a:

hot drink ☐ **cold drink?** ☐

If so, which type(s).....

(iii) What does your child generally use for drinking:

bottle yes☐ **no** ☐ **feeder cup yes**☐ **no** ☐ **cup? yes**☐ **no** ☐

(iv) Does your child have a <u>snack</u> during the day? **yes**☐ **no** ☐

If yes, on how many occasions per day? ☐

Which snacks are given:

biscuits yes☐ **no** ☐ **sweets yes**☐ **no** ☐ **crisps yes**☐ **no** ☐ **fruit yes**☐ **no** ☐

other?.....(please specify)

(v) Which of these does your child have at <u>bedtime:</u>

drink yes☐ **no** ☐ **snack yes**☐ **no** ☐

(vi) Which of these does your child have <u>during the night:</u>

drink yes☐ **no** ☐ **snack yes**☐ **no** ☐

Q3 (a) Are your child's teeth brushed? **yes**☐ **no** ☐

(b) If yes, how many times per day? ☐

(c) By whom? **parent yes**☐ **no** ☐ **child yes**☐ **no** ☐ **other? Yes** ☐ **no** ☐

(d) Is toothpaste used? **yes**☐ **no** ☐

(e) Do you give your child:

fluoride tablets yes☐ **no** ☐ **fluoride drops daily yes**☐ **no** ☐

Q4 (i) Does your child attend **<u>day-care/nursery</u>** more than twice per week: **yes**☐ **no** ☐

If yes, please name facility .....

(ii) Is your child in the care of a **<u>relative/childminder</u>** more than two half-days / evenings per week: **yes**☐ **no** ☐

Q5 When you carry out the **bulk** of your food shopping, how do you travel **<u>to</u>** the shops?

on foot☐**1** **by bus**☐**2** **by car**☐**3** **by taxi**☐**4**

THANK YOU for your help with this study

Study Number ☐☐☐☐☐☐

HEALTH VISITOR / MUTANS STUDY

CONFIDENTIAL **PARENTAL QUESTIONNAIRE (4 YEARS)**

Thank you again for your co-operation in this study. Please answer the following questions as accurately as possible and return the form to the Health Visitor. All information is completely confidential and for research purposes only. To be completed by parent / guardian.

Today's date: ☐☐**dd** ☐☐**mm** ☐☐**yy**

Q1 (i) Which meals has your child each day: **breakfast yes**☐ **no** ☐ **dinner yes**☐ **no** ☐

tea yes☐ **no** ☐ **supper yes**☐ **no** ☐

(ii) Between meals, in general, how many times per day does your child have a:

hot drink ☐ **cold drink?** ☐

If so, which type(s) .....

(iii) What does your child generally use for drinking:

bottle yes☐ **no** ☐ **feeder cup yes**☐ **no** ☐ **cup? yes**☐ **no** ☐

(iv) Does your child have a snack during the day? **yes**☐ **no** ☐

If yes, on how many occasions per day? ☐

Which snacks are given:

biscuits yes☐ **no** ☐ **sweets yes**☐ **no** ☐ **crisps yes**☐ **no** ☐ **fruit yes**☐ **no** ☐

other? .....(please specify)

(v) Which of these does your child have at bedtime:

drink yes☐ **no** ☐ **snack yes**☐ **no** ☐

(vi) Which of these does your child have during the night:

drink yes☐ **no** ☐ **snack yes**☐ **no** ☐

Q2 (a) Are your child's teeth brushed? **yes**☐ **no** ☐

(b) If yes, how many times per day? ☐

(c) By whom? **parent yes**☐ **no** ☐ **child yes**☐ **no** ☐ **other? yes**☐ **no** ☐

(d) Is toothpaste used? **yes**☐ **no** ☐

(e) Do you give your child: **fluoride tablets yes**☐ **no** ☐ **fluoride drops daily yes**☐ **no** ☐

Q3 (i) Does your child attend **day-care/nursery** more than twice per week: **yes**☐ **no** ☐

If yes, please name facility .....

(ii) Is your child in the care of a **relative/childminder** more than two half-days / evenings per week: **yes**☐ **no** ☐

Q4 When you carry out the **bulk** of your food shopping, how do you travel **to** the shops?

on foot☐**1** **by bus**☐**2** **by car**☐**3** **by taxi**☐**4**

Q5 Please look at the following list of qualifications: starting from number ☐ please tick the first one you come to which the **MOTHER** (of the child in the study) has passed:

☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
Degree	**'A' Level or SCE Higher**	**'O' Level passes (Grades A-C if after 1975) or Standard Grade (Levels 1-3)**	**CSE Grades 2-5 or Standard Grade (level 4,5)**	**No formal qualifications**

If qualifications not listed, please specify: .....

THANK YOU for your help with this study

3.7 Health visitor questionnaires for each of the four years of the study

HEALTH VISITOR/MUTANS STUDY

CONFIDENTIAL

Study No.

SOCIAL / MEDICAL / ORAL INFORMATION SHEET

(12 months)

To be completed by Health Visitor

date: / /

Child's study no. | Health Visitor's name:

Number of teeth present at 1 year | Health Visitor's Assessment: Is the child at high risk of developing dental decay? yes/no

Weight at birth | Weight at 8 weeks | Centile | Weight at 8 months | Centile

Height at birth | Height at 8 weeks | Height at 8 months

Head Circumference at birth (O.F.C.) | O.F.C. at 8 weeks | O.F.C. at 8 months

Immunisation status ..... Ethnic origin .....

Pregnancy, Delivery and Neo-natal data of significance .....

.....

Does the child suffer from any illness(es) requiring long-term medication? yes/no

If yes, nature of illness and medication .....

.....

Age at weaning to solids? ..... months Has the child been breast fed? yes/no

Use of dummy/comforter? yes/no. If yes, still being used? yes/no Are vitamin supplements given? yes/no

Feeding problems of significance? .....

.....

Number of siblings? Birth Order

Mother's d.o.b. / / Married /Single/Co-habiting/Living with parents

Mother's employment: no/part-time/full-time Nature of employment: .....

Father's employment/past employment .....

Parents Health - any significant data?

Mother: ....Smoker/Non-Smoker

Father: .....Smoker/Non-Smoker

Housing: Owner/Occupier Private Rent Council/SSHA Rent

If you have any relevant additional information please add this overleaf.

Study Number

HEALTH VISITOR/MUTANS STUDY

CONFIDENTIAL

SOCIAL / MEDICAL / ORAL INFORMATION SHEET

(2 YEARS)

To be completed by Health Visitor

Today's date: ☐dd ☐mm ☐yy

Health Visitor's name: ..... Sample taken: At home ☐ At clinic ☐ Other ☐

eg. nursery, childminder etc

Number of teeth present at 2 years? ☐

Health Visitor's Assessment : Is the child at high risk of developing dental decay? yes☐ no ☐

Weight at 2 years ☐☐ Centile ☐ Height at 2 years ☐☐ Centile ☐ O.F.C. at 2 years ☐☐☐

Immunisation status? Complete: yes☐ no ☐ Incomplete: yes☐ no ☐

Does child suffer from any illness(es) requiring long term medication? yes☐ no ☐

If yes, nature of illness and medication: .....

.....

Use of dummy/comforter? yes☐ no ☐ If yes, still being used? yes☐ no ☐

Are vitamin supplements given? yes☐ no ☐

Feeding problems of significance? .....

Number of siblings? ☐

Mother's employment : part-time: ☐ full-time: ☐ unemployed: ☐ none: ☐ Nature of employment: .....

Mother married: ☐ single: ☐ cohabiting: ☐ living with parents? ☐

Father's employment : part-time: ☐ full-time: ☐ unemployed: ☐ none: ☐ Nature of employment: .....

Parents Health - any significant data?

Mother: ..... Smoker? yes☐ no ☐

Father: ..... Smoker? yes☐ no ☐

Housing : Owner/Occupier: yes☐ no ☐ Private Rent: yes☐ no ☐ Council/SSHA Rent: yes☐ no ☐

Updated address

If moved in last 12 months :

Postcode:

If you have any relevant additional information please add this overleaf.

Study Number

HEALTH VISITOR/MUTANS STUDY

CONFIDENTIAL

SOCIAL / MEDICAL / ORAL INFORMATION SHEET
(3 YEARS)

To be completed by Health Visitor

Today's date: ☐dd ☐mm ☐yy

Health Visitor's name: ..... Sample taken: At home ☐ At clinic ☐ Other ☐

eg. nursery, childminder etc

Number of teeth present at 3 years? ☐

Health Visitor's Assessment : Is the child at high risk of developing dental decay? yes☐ no ☐

Immunisation status? Complete: yes☐ no ☐

Does child suffer from any illness(es) requiring long term medication? yes☐ no ☐

If yes, nature of illness and medication: .....

.....

Use of dummy/comforter? yes☐ no ☐ If yes, still being used? yes☐ no ☐

Are vitamin supplements given? yes☐ no ☐

Feeding problems of significance? .....

Number of siblings? ☐

Mother's employment : part-time: ☐ full-time: ☐ unemployed: ☐ none: ☐ Nature of employment: .....

Mother married: ☐ single: ☐ cohabiting: ☐ living with parents? ☐

Father's employment : part-time: ☐ full-time: ☐ unemployed: ☐ none: ☐ Nature of employment: .....

Parents Health - any significant data?

Mother: ..... Smoker? yes☐ no ☐

Father: ..... Smoker? yes☐ no ☐

Housing : Owner/Occupier: yes☐ no ☐ Private Rent: yes☐ no ☐ Council/SSHA Rent: yes☐ no ☐

Updated address

If moved in last 12 months :

Postcode: Telephone:

If you have any relevant additional information please add this overleaf.

Study Number

HEALTH VISITOR/MUTANS STUDY

CONFIDENTIAL

SOCIAL / MEDICAL / ORAL INFORMATION SHEET

(4 YEARS)

To be completed by Health Visitor

Today's date: ☐dd ☐mm ☐yy

Health Visitor's name: ..... Sample taken: At home ☐ At clinic ☐ Other ☐

eg. nursery, childminder etc

Number of teeth present at 4 years? ☐

Weight at 3½ years ☐☐ Centile

Height at 3½ years ☐☐ Centile

Health Visitor's Assessment : Is the child at high risk of developing dental decay? yes☐ no ☐

Is the child registered with a dentist yes☐ no ☐

Immunisation status? Complete: yes☐ no ☐

Does child suffer from any illness(es) requiring long term medication? yes☐ no ☐

If yes, nature of illness and medication: .....

.....

Use of dummy/comforter? yes☐ no ☐ If yes, still being used? yes☐ no ☐

Are vitamin supplements given? yes☐ no ☐

Feeding problems of significance? .....

Number of siblings? ☐

Mother's employment : part-time: ☐ full-time: ☐ unemployed: ☐ none: ☐ Nature of employment: .....

Mother married: ☐ single: ☐ cohabiting: ☐ living with parents? ☐

Father's employment : part-time: ☐ full-time: ☐ unemployed: ☐ none: ☐ Nature of employment: .....

Parents Health - any significant data?

Mother: ..... Smoker? yes☐ no ☐

Father: ..... Smoker? yes☐ no ☐

Housing : Owner/Occupier: yes☐ no ☐ Private Rent: yes☐ no ☐ Council/SSHA Rent: yes☐ no ☐

Updated address

If moved in last 12 months :

Postcode: Telephone:

If you have any relevant additional information please add this overleaf.

3.8 Health visitor feedback questionnaires for years 2, 3 and 4 of the study

Health Visitor/ Mutans Study Questionnaire 2

This questionnaire is to obtain feedback and constructive criticism in order to monitor progress of the study.

Date ☐☐☐

Please tick one box only for each question.

1. Do you find the **sampling information** (labels etc.) sent to you
   - very satisfactory ☐ 1
   - satisfactory ☐ 2
   - unsatisfactory ☐ 3
   - very unsatisfactory? ☐ 4

2. Do you find **planning for sampling visits**
   - easy ☐ 1
   - fairly easy ☐ 2
   - difficult ☐ 3
   - very difficult? ☐ 4

3. Within your timetable, is the **time available for sampling**
   - adequate ☐ 1
   - fairly adequate ☐ 2
   - inadequate ☐ 3
   - very inadequate? ☐ 4

4. Do you find the **distribution** and **number** of cool-bags
   - very satisfactory ☐ 1
   - satisfactory ☐ 2
   - unsatisfactory ☐ 3
   - very unsatisfactory? ☐ 4

5. Do you find the sample **collection arrangements**
   - very satisfactory ☐ 1
   - satisfactory ☐ 2
   - unsatisfactory ☐ 3
   - very unsatisfactory? ☐ 4

6. Did you find the **sampling procedure for the *2* year olds**
   - very easy ☐ 1
   - easy ☐ 2
   - a little difficult ☐ 3
   - very difficult? ☐ 4

7. Did the parents, in general, find the ***2* year parental questionnaire**

very simple ☐ 1
simple ☐ 2
a little hard ☐ 3
very hard? ☐ 4

8. Did you find completion of the ***2* year HV questionnaire**

very easy ☐ 1
easy ☐ 2
difficult ☐ 3
very difficult? ☐ 4

9. Was the ***2* year** HV questionnaire **easier** than the ***1* year** HV questionnaire?

Yes ☐ 1
No ☐ 2
No Different ☐ 3

10. Is the ***3* year** HV questionnaire **easier** than the ***2* year** HV questionnaire?

Yes ☐
No ☐
No Different ☐

11. In general, can you foresee any **major hurdles** during the remainder of the study?

Yes ☐ 1
No ☐ 2

If yes, what?.....

.....

.....

12. Are there any other comments you would like to make regarding the study?

(Please continue below if necessary) .....

.....

.....

13. How do you rate Dental Health as a priority for Health Visitors ☐

(Scale 0 - 10, where 0 = no priority)

Thank you for your continuing help with this study.

Health Visitor/ Mutans Study Questionnaire 3

This questionnaire is to obtain feedback and constructive criticism in order to monitor progress of the study.

Date ☐☐☐

Please tick one box only for each question.

1. Do you find the **sampling information** (labels etc.) sent to you
   - very satisfactory ☐ 1
   - satisfactory ☐ 2
   - unsatisfactory ☐ 3
   - very unsatisfactory? ☐ 4

2. Do you find **planning for sampling visits**
   - easy ☐ 1
   - fairly easy ☐ 2
   - difficult ☐ 3
   - very difficult? ☐ 4

3. Within your timetable, is the **time available for sampling**
   - adequate ☐ 1
   - fairly adequate ☐ 2
   - inadequate ☐ 3
   - very inadequate? ☐ 4

4. Do you find the **distribution** and **number** of cool-bags
   - very satisfactory ☐ 1
   - satisfactory ☐ 2
   - unsatisfactory ☐ 3
   - very unsatisfactory? ☐ 4

5. Do you find the sample **collection arrangements**
   - very satisfactory ☐ 1
   - satisfactory ☐ 2
   - unsatisfactory ☐ 3
   - very unsatisfactory? ☐ 4

6. Did you find the **sampling procedure for the *3* year olds**
   - very easy ☐ 1
   - easy ☐ 2
   - a little difficult ☐ 3
   - very difficult? ☐ 4

7. Did the parents, in general, find the ***3* year parental questionnaire**

very simple ☐ 1
simple ☐ 2
a little hard ☐ 3
very hard? ☐ 4

8. Did you find completion of the ***3* year HV questionnaire**

very easy ☐ 1
easy ☐ 2
difficult ☐ 3
very difficult? ☐ 4

9. Is the ***4* year** HV questionnaire **easier** than the ***3* year** HV questionnaire?

Yes ☐ 1
No ☐ 2
No Different ☐ 3

10. In general, can you foresee any **major hurdles** during the remainder of the study?

Yes ☐ 1
No ☐ 2

If yes, what?.....

.....

.....

11. Are there any other comments you would like to make regarding the study?

(Please continue below if necessary) .....

.....

.....

12. How do you rate Dental Health as a priority for Health Visitors ☐

(Scale 0 - 10, where 0 = no priority)

Thank you for your continuing help with this study.

Health Visitor/ Mutans Study Questionnaire 4

This questionnaire is to obtain feedback and constructive criticism in order to monitor progress of the study.

Date ☐☐☐

Please tick one box only for each question.

1. Do you find the **sampling information** (labels etc.) sent to you
   - very satisfactory ☐ 1
   - satisfactory ☐ 2
   - unsatisfactory ☐ 3
   - very unsatisfactory? ☐ 4

2. Do you find **planning for sampling visits**
   - easy ☐ 1
   - fairly easy ☐ 2
   - difficult ☐ 3
   - very difficult? ☐ 4

3. Within your timetable, is the **time available for sampling**
   - adequate ☐ 1
   - fairly adequate ☐ 2
   - inadequate ☐ 3
   - very inadequate? ☐ 4

4. Do you find the **distribution** and **number** of cool-bags
   - very satisfactory ☐ 1
   - satisfactory ☐ 2
   - unsatisfactory ☐ 3
   - very unsatisfactory? ☐ 4

5. Do you find the sample **collection arrangements**
   - very satisfactory ☐ 1
   - satisfactory ☐ 2
   - unsatisfactory ☐ 3
   - very unsatisfactory? ☐ 4

6. Did you find the **sampling procedure for the *4* year olds**
   - very easy ☐ 1
   - easy ☐ 2
   - a little difficult ☐ 3
   - very difficult? ☐ 4

7. Did the parents, in general, find the ***4*** **year parental questionnaire**

very simple ☐ 1
simple ☐ 2
a little hard ☐ 3
very hard? ☐ 4

8. Did you find completion of the ***4*****year HV questionnaire**

very easy ☐ 1
easy ☐ 2
difficult ☐ 3
very difficult? ☐ 4

9. Is the ***4*** **year** HV questionnaire **easier** than the ***3*** **year** HV questionnaire?

Yes ☐ 1
No ☐ 2
No Different ☐ 3

10. Are there any other comments you would like to make regarding the study?

(Please continue below if necessary) .....

.....

.....

11. Do you have any suggestions on how we could take this study forward (given that we can identify high-risk infants).

12. How do you rate Dental Health as a priority for Health Visitors ☐

(Scale 0 - 10, where 0 = no priority)

Thank you for your continuing help with this study.

feedbck4\1

3.9 Information leaflet on study issued to health visitors

University
Of Dundee
DENTAL SCHOOL

Health Visitor/Mutans Study

Information Leaflet

iii) Collection and Drop-off Points

Ryehill Health Centre

Wallacetown Health Centre

Broughty Ferry Health Centre

West Gate Health Centre - this location has recently been added to service the west - end of Dundee

All samples should be delivered to one of these locations by 16:30 hours. If you anticipate being late for the pick up then please call Heather Ballantyne at the Dental Hospital Tel: (0382) 26041 ext 232 or (08501) 30841 (mobile)

Finally, do give me a call if you are unhappy with any of these arrangements.

Heather .M. Ballantyne.

I Trigger Letters:

A few weeks before each study child's birthday you will receive a list of your study children for the following month. Together with this list will be stamped addressed envelopes containing two letters to the parents who have agreed that their children can participaté in the study. The letters (one printed on white sheet and the other on green sheet) are virtually identical - they contain

1) confirmation for the parents that they have agreed their child can take part in the study.

2) notice that you will be contacting them soon to arrange a visit to carry out the saliva sampling and completion of the questionnaire, and

3) advising them that the dental examination of their child will take place at this or a separate visit. The white sheet suggests a home visit, the green sheet a clinic/surgery visit. You will decide which of the letters is more convenient for that household.

II Visits:

You are then asked to arrange the visit with the parents/guardians (or carers) within one month of the first birthday of the study child.

Once a visit has been arranged for the child, you should contact me, Heather Ballantyne, or if I am unavailable, Chris Longbottom or John Radford - Tel: (0382) 26041 ext 232 or (08501) 30841 (mobile) - so that I can arrange a visit at the same time. If I am too busy I will contact the parents direct to arrange my visit.

III Questionnaires:

You will be sent a set of Parental Questionnaires (yellow) and Social/Medical/Oral Information Sheets (white) for completion. Return envelopes will also be provided. At each Child's visit you should ask the parent/guardian to complete the parental questionnaire and place it in the addressed envelope. Please,

wherever possible return the completed parental questionnaire to the Health Centre box at the same time as the samples are dropped into the igloo. At the sampling visit you should note the number of teeth the child has present and assess whether the child is at high risk of developing dental decay. At about this time you should complete the Social/Medical/Oral Information Sheet for each child and send it in a 'Health visitor/Mutans Study' envelope through the internal mail or drop it into one of the Health Centre boxes

Stamped addressed envelopes will be provided for those parental questionnaires which need to be given to a carer to pass on to the parent for completion.

IV Saliva Sample:

i) Contents of Cool-bags

Extra labels: in case printed labels are unavailable

Pen: for marking labels if necessary

Sampling loops: approximately 5 in sterile packets

Sampling vials: approximately 5 containing transport medium

Storage box: expanded polystyrene containing approximately 5 vials

As a reminder, there is a simple guide to sampling on the lid.

ii) Sampling

Before sampling:

a) Each child has been given a 10 digit study number to be used for the entire duration of the study. With the trigger letter you will be sent 3 labels for each child printed with the study number - one for the sample vial, one for the parental questionnaire, and one for the Social/Medical/Oral information sheet.

b) Pick up a cool-bag from igloo marked 'FRESH' situated in the reception office of your Health Centre.

This can be stored in a fridge for up to one week. If kept longer the transport medium in the vial will be useless.

Taking Sample:

1) Label vial - use printed or hand written label

2) Remove sterile loop from wrapper

3) Remove vial from storage box and open

4) Draw loop across top surface of tongue completely filling loop with saliva and tongue debris.

5) Dislodge material from loop into vial by agitation.

6) Close vial

7) Dispose of loop into household waste

8) Place vial in storage box, checking label

9) Return to cool-bag

10) Return to igloo marked 'USED'

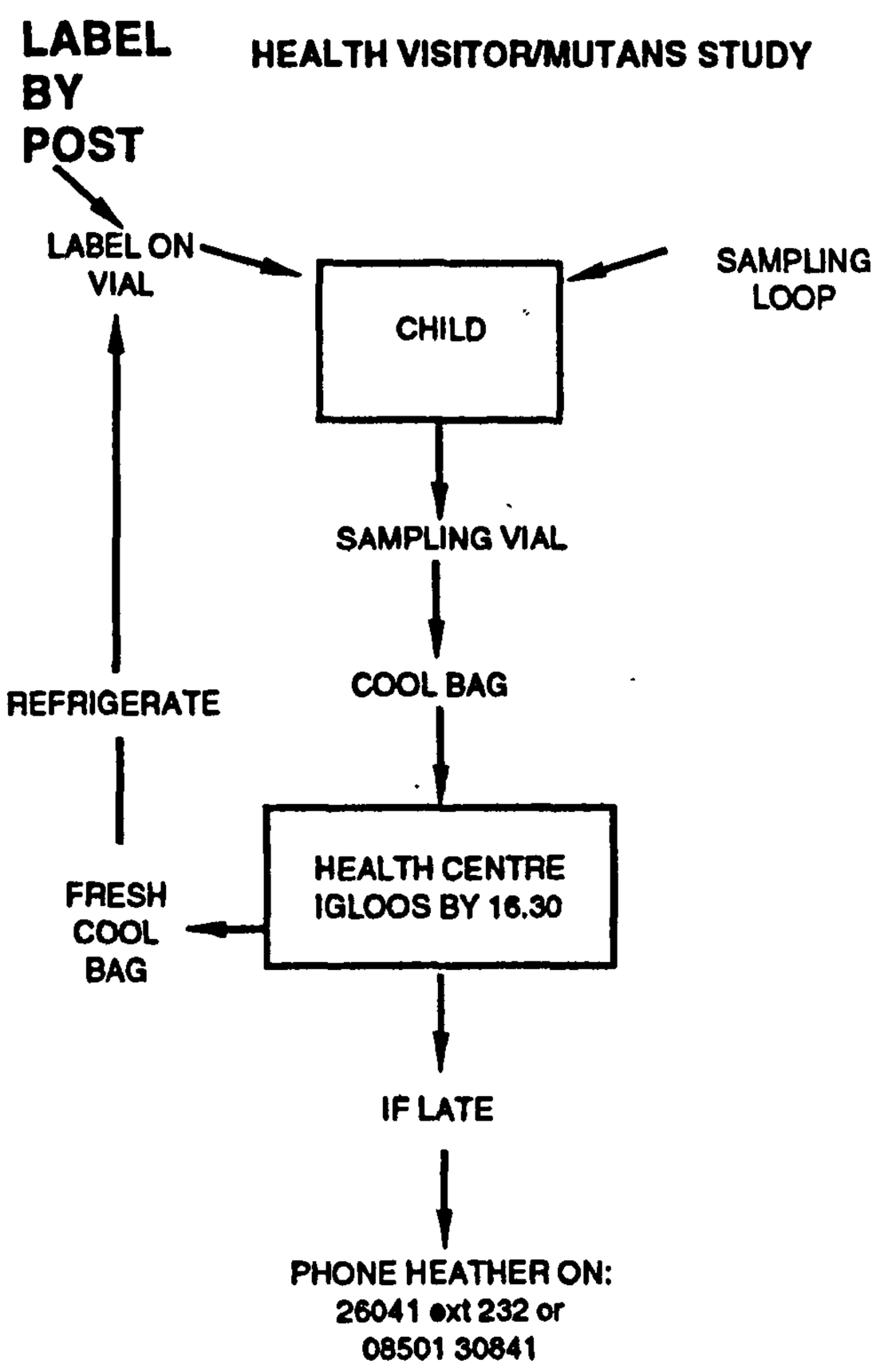
11) Pick up a fresh cool-bag from igloo if sampling again within one week.

Do not worry at all if many of the vials have not been used - we will replace them with fresh ones

Do not keep samples overnight

Do return all samples taken that day

3.10 Flow diagram on study methodology issued to health visitors


LABEL BY POST
HEALTH VISITOR/MUTANS STUDY
LABEL ON VIAL
CHILD
SAMPLING LOOP
SAMPLING VIAL
COOL BAG
REFRIGERATE
HEALTH CENTRE IGLOOS BY 16.30
FRESH COOL BAG
IF LATE
PHONE HEATHER ON:
26041 ext 232 or
08501 30841

3.11 Examples of newsletters issued to health visitors

Feedback questionnaire

Thank you all for your replies to the feedback questionnaire. We hope you all enjoyed the buffet lunch at Wallacetown in May.

The main points raised were :

- More locations for igloos required - we hope that new igloos at Douglas Clinic will help

- Many of you are worried about increasing numbers of parents returning to work resulting in reduced time to visit. This will be an increasing problem but it may be positive in that several infants can be accessed simultaneously in nurseries

Looking ahead

- Vitally important that we try and catch these children that have been missed - these will probably be the ones with decay and are crucial to the study!

- Unfortunately, these good results for the first year are not reflected in the two year olds to date - any feedback would be appreciated (Use enclosed comments slip)

- Heather will now dentally examine the mothers of the 40 one year olds with decay and 40 random controls

- Two papers at present being finalised for publication, one for the Journal of Community Dentistry and Oral Epidemiology, the other for the Journal of the Health Visitors Association

- Oral presentation to be done at the health visitor's conference in May 1996

- Second oral and poster presentations at the conference for the British Dental Society for Dental Research in April 1996

- We hope to have a repeat 'get-together' in the future, in addition to newsletters in order to keep you updated on the study

Thank you for your co-operation

HV/MUTANS STUDY NEWSLETTER

No. 1 **November 1995**

This is the first of bi-monthly newsletters aimed at updating you on the progress of the study.

First of all a big 'THANK YOU ' for all your hard work. The results from the first year are encouraging in terms of the success of the study itself, but unfortunately reflect that decay is present in the 1 year olds already - not such an encouraging sign for the dental health of Tayside children.

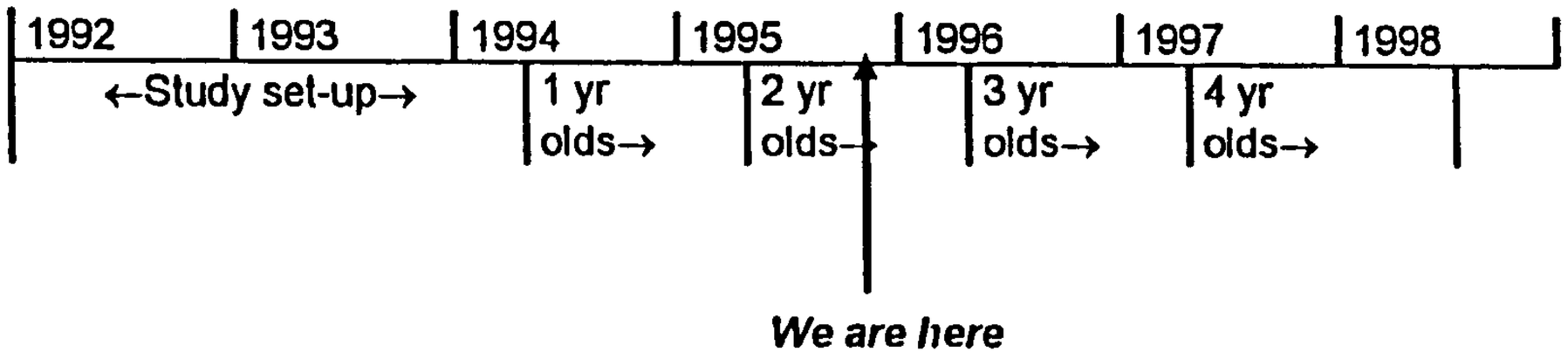
What are our aims?

Firstly, we are looking for ways of predicting decay in pre-school children before it occurs. This is the reason for following all infants through to 4 years old, identifying which have decay and then looking retrospectively at the factors which have caused it. If decay can be predicted at an early age, those individuals could then be targeted with preventive techniques.
Secondly, and of equal importance, we assessing the feasibility of being able to access pre-school infants through health visitors with the aim that in the future this may provide an opportunity for assessment to be made of the caries-risk. These infants at highest risk could therefore be treated and/or referred.
This is one of the reasons it is vitally important for the sample to be taken by a health visitor. This is the only way we can assess the feasibility of this procedure being carried out in the future as part of the overall developmental screening process.

How can decay be predicted?

We will look specifically at microbiological, socio-economic, medical and dental factors. The saliva samples give us information on which types and numbers of bacteria are in the mouth - specifically mutans streptococci which is the main 'bug' causing decay. The parental and the HV questionnaires provide the socio - economic and medical data and the dental examination determines the condition of the teeth.

Where are we now?



What are the results for the 1 year olds?

Study cohort - all infants born between 1 April 1993 and 31 March 1994

Of the **1974** infants born **1747** were consented to participate, a consent rate of **88.5%**

Of the 1747 infants consented

Number of saliva samples taken by the health visitor **1440**

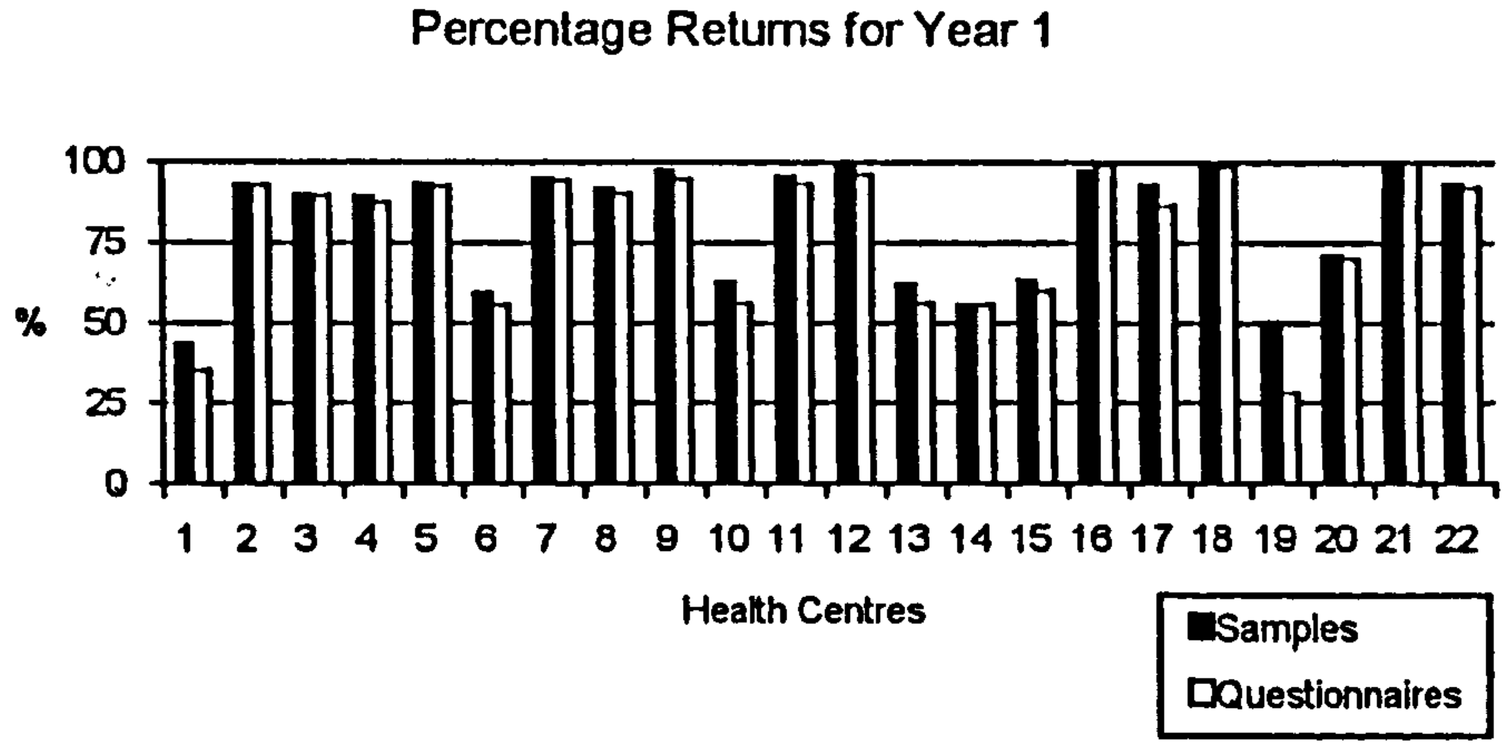
Number of questionnaires completed **1383 (HV) & 1372 (P)**

Number of dental examinations completed **1408**

Number of infants with dental decay **41**

This gives a running total of **1372** out of the **1747** consented **(78.5%)** for whom all procedures have been completed

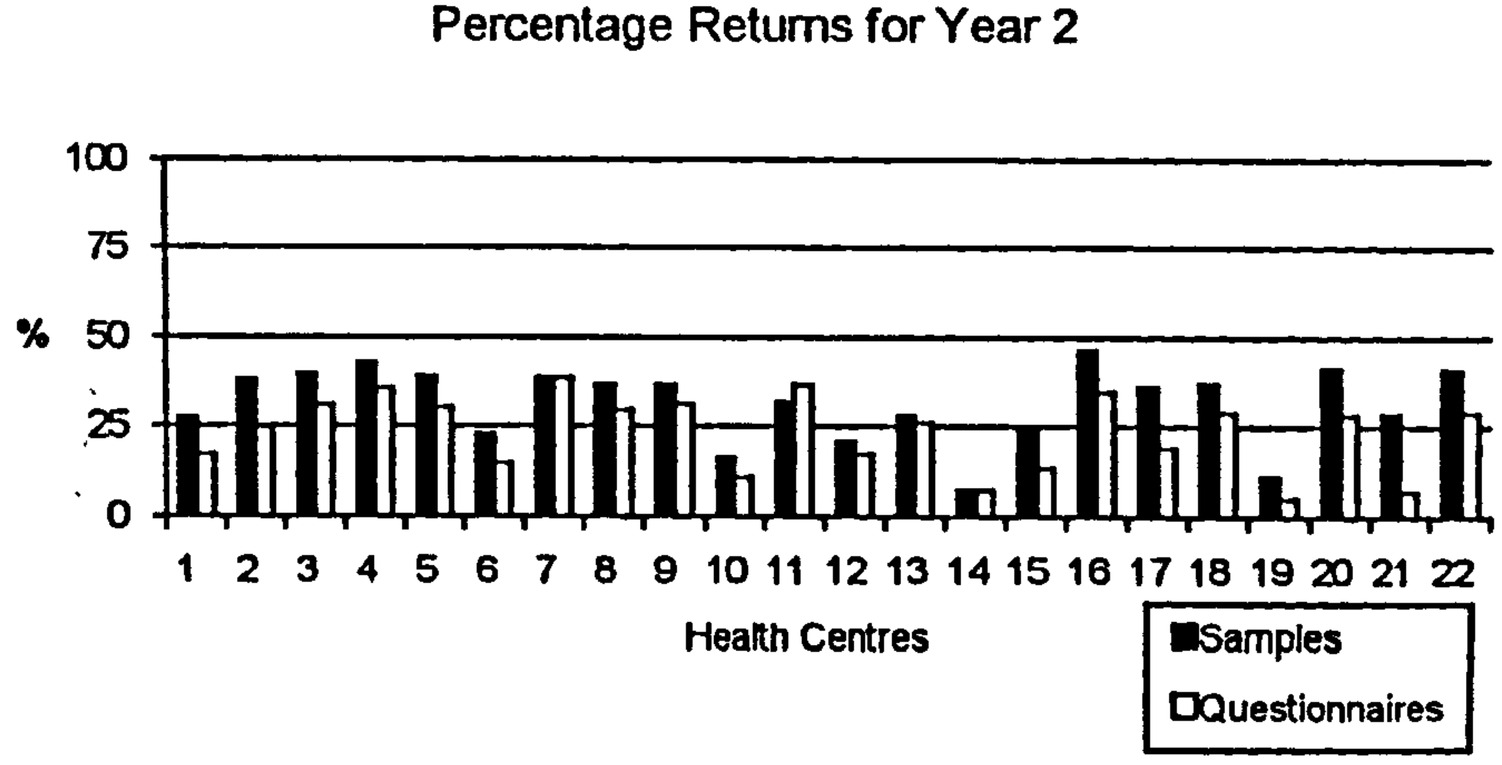
Comparative breakdown of health centres



Main conclusions from 1st year results

- good consent rate **88.5%**
- still require HV questionnaires **364**
- still require parental questionnaires **375**
- still require saliva samples **307** *(Heather cannot examine consented infants until they have been sampled)*
- still require dental examination **339**

(Lists and your Health Centres' confidential position on the graph are provided)



Comment : Year 2 returns show large change from Year 1

No. 5 December 1996

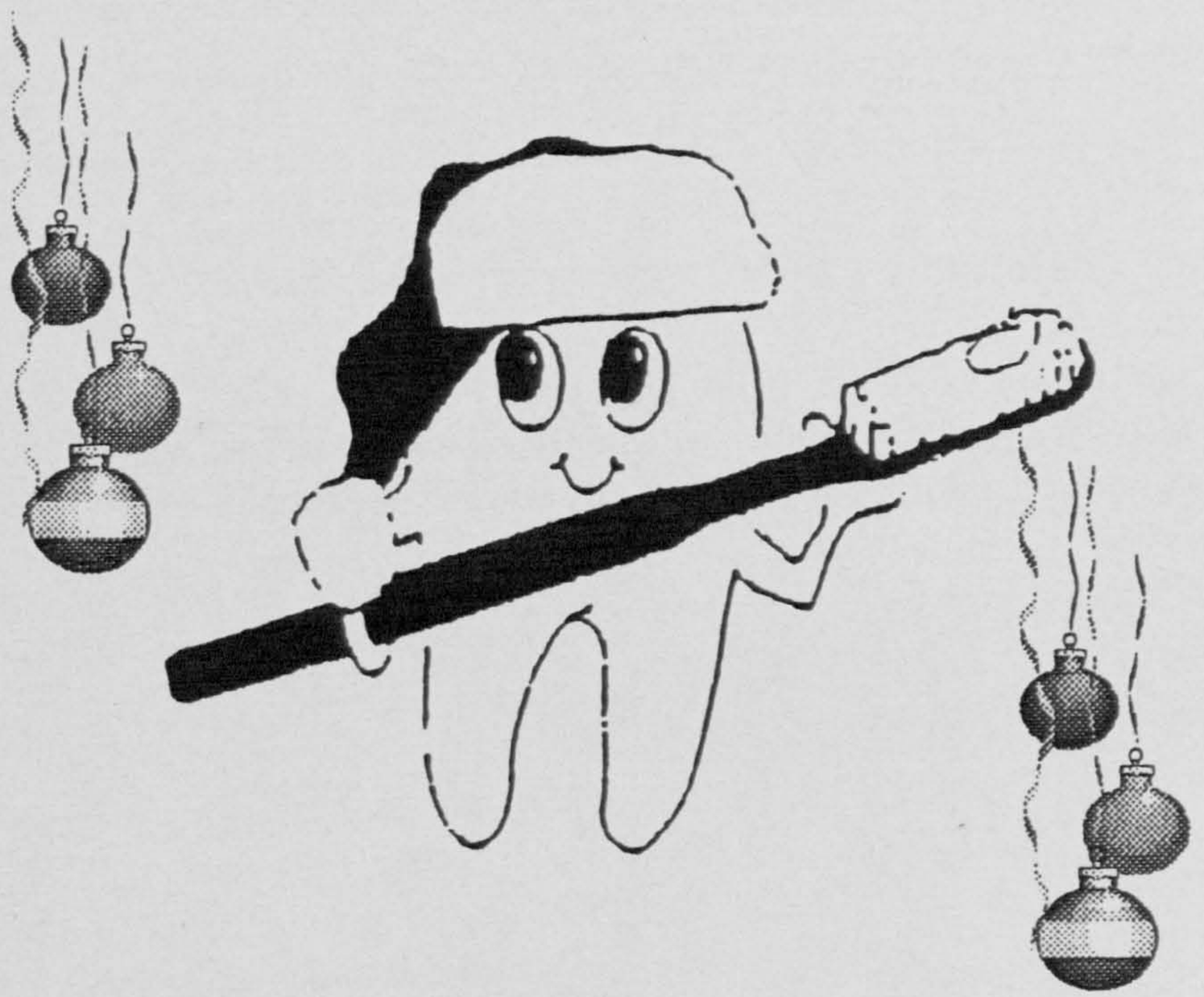
HV / MUTANS STUDY NEWSLETTER

Finally

The good news is, that as of April we will be into the final year of the study. Our aim is to have international recognition and be able to praise the hard work of all health visitors and nursing staff involved, (and don't forget the raffle for the 22 cool-boxes used in the study!).

We would like to take this opportunity to send you our good wishes for the festive season.

A Very Merry Christmas and Best Wishes for 1997.



Thank you again for all your help with this study.

Printed by the Mutan Study Team

Feedback Questionnaires

Thank you for returning these questionnaires. We are still however missing 18 (29%) out of a total of 62. If any one requires a replacement ***feedback questionnaire*** please let us know and we will be happy to post one out.

The information collected from these questionnaires helps us to pin-point potential hurdles and ensures the study finishes successfully and on time.

Igloos at Douglas Clinic

Can we remind everyone of the igloos at Douglas Clinic. These were placed to increase availability of sampling kits in the east end of the city but at present are not as frequently used as expected.

Remember that this is the first pick-up point by the taxis' and samples need to be in the igloos at Douglas by 4.00 pm.

Missing data

A big thank you to all those who have put an incredible effort into catching up with as much of the 2 year old data as possible. We are unfortunately still missing 313 HV questionnaires and 351 parental questionnaires but are in the process of collating individual lists for everyone.

Joint visits

Please do keep informing Heather of the dates and times of your visits to the study children and she will visit at the same time if at all possible.

Festive Period

As usual we will be ***temporarily suspending*** sampling for the holiday period from Monday 23rd December until Friday 3rd January inclusive.

Please feel free to send in any questionnaires during this period and Heather and Maureen will be around at intervals to answer any queries and problems as soon as possible.

To finish

- Remember we have had a new telephone number since January (01382 345755 / 345751).
- Let us know if you have any comments / queries / problems
- A big thanks from Heather to those who have helped arrange clinics to reduce the number of home visits
- We'll let you know soon regarding the finalised date for the results seminar

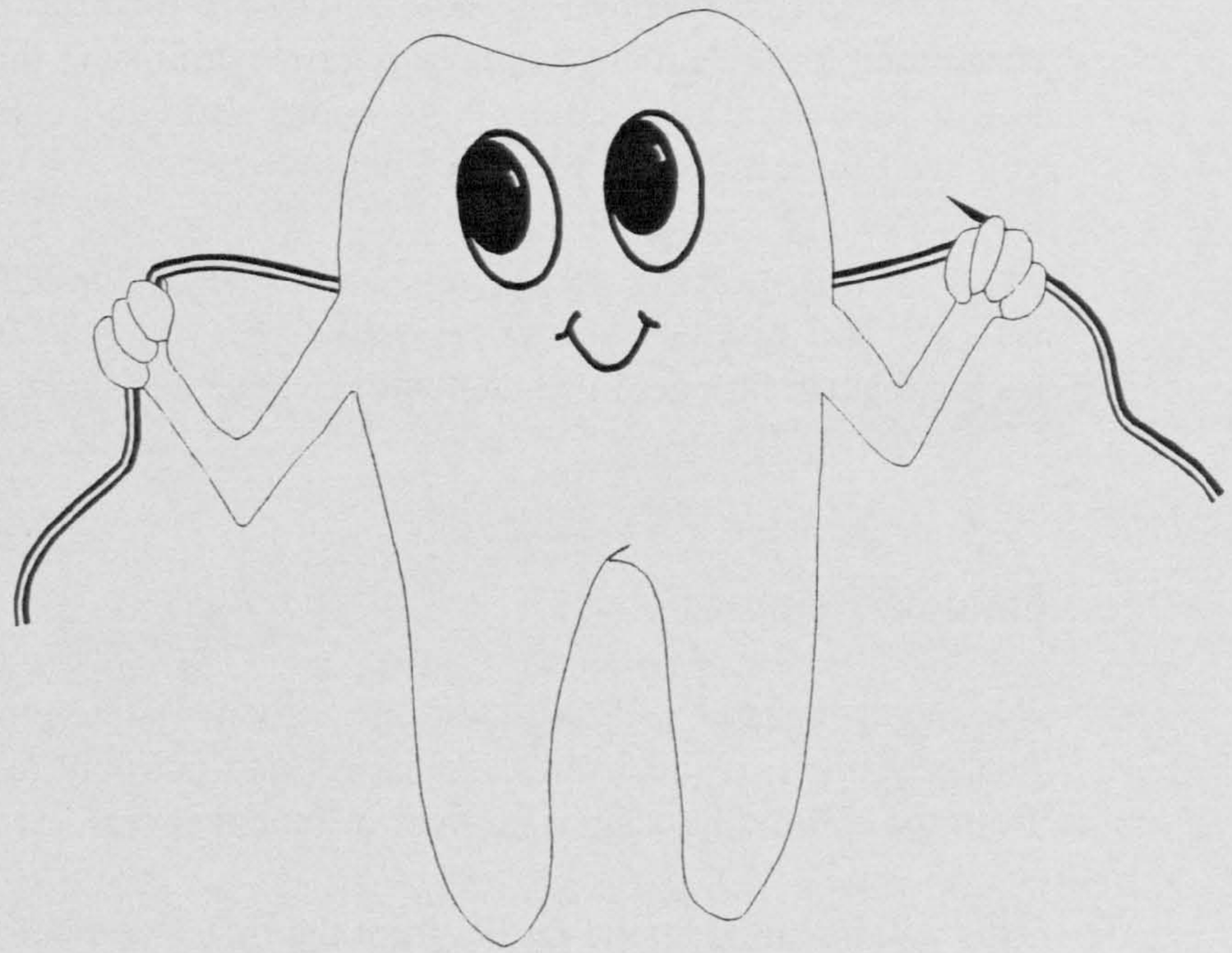
Carry on sampling. We'll be finished this time next year!

Thank you all for your continued help and support

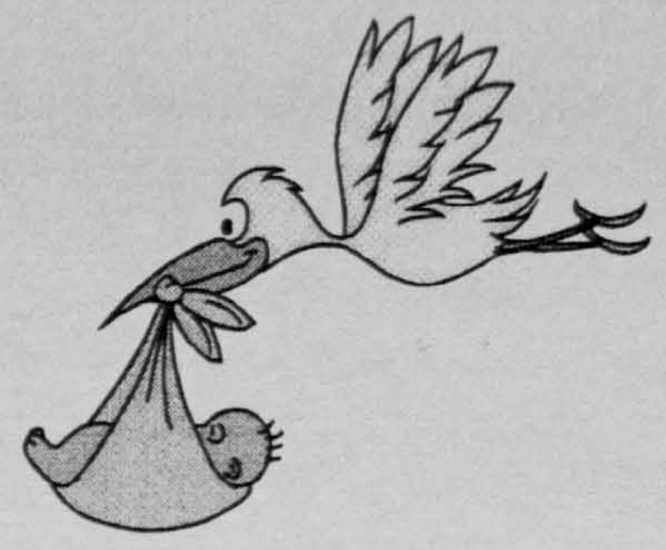
No. 7

HV / MUTANS STUDY NEWSLETTER

May 1997



Congratulations to Heather



As most of you will now know, Heather is having a baby in August **but the study goes on**!!

All plans will be revealed as soon as they are finalised but Heather is continuing to do as many visits as possible until such times as it is no longer feasible. This includes both home visits and clinic visits so if you need some company please let her know.

There is still quite a backlog of 3 year olds in the study for whom samples and questionnaires are needed. If you have any problems seeing them please get in contact with Heather or Maureen.

Results Seminar

We intend to hold a "results seminar" (food will be provided) for all health visitors, clinical nurse managers and possibly a representative from the community dental services in June this year.

This will be based on the results from the first 3 years of the study with a brief look at how the fourth and final year is progressing. Unfortunately at present we are really short of 3 year old data and would be really grateful if we could have as much of this in before the seminar so we can do the appropriate statistics and provide you with the best possible results.

Enclosed in the envelope is an individual list of 3 year old samples and questionnaires which are missing for your practice. Don't worry if you've sent them in the last week or so, they will not have been entered onto the computer as yet! We also hope to produce a graph again to see how everyone is doing and will present this at the results seminar.

We'd just like to say a big thank you again for all your hard work and remind you that we are in the last year of the study so 'hang in there'!

Sample Bags

A desperate plea from the study technician for any silver sample bags lurking in fridges, desk drawers, car boots etc.!. We now have only the bare minimum to keep the igloos packed every day.

If you have any could you please place them in the nearest 'used' igloo or hand them into the Dental Hospital. It would cost about £100.00 to buy just 25 more and Heather is striving hard to maintain the study within budget or facing a docking of wages (joke)! Your help would be appreciated.

Dental examinations

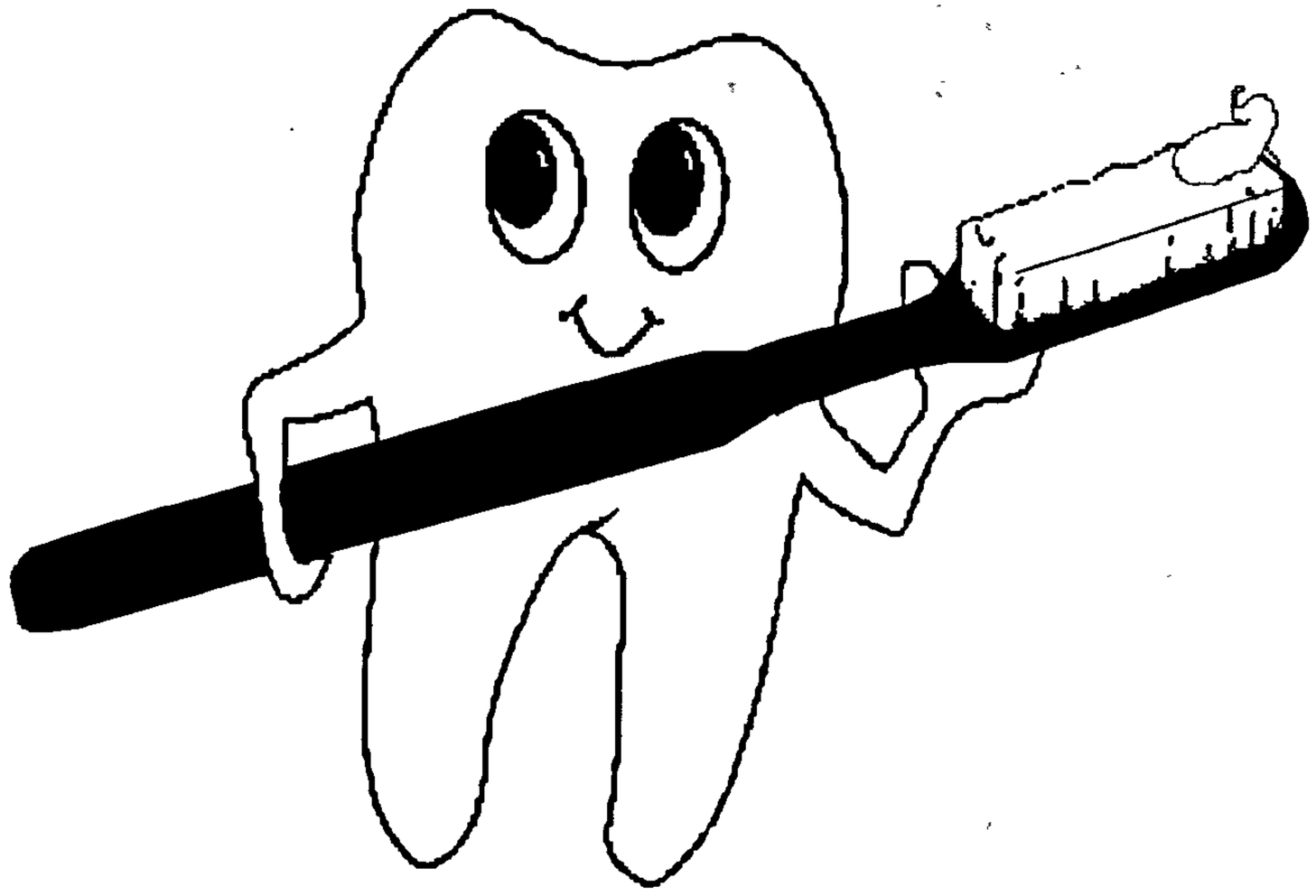
Just a reminder to let the mums know that Heather will still be visiting each child to examine their teeth if she wasn't present when the saliva sample was taken.

Several mums have said to Heather that the health visitor had already been to do everything for the study and didn't expect to have someone else at the door! This would be much appreciated as it has caused a wee bit of confusion even although Heather always sends a letter before she visits. Thanks for your help.

No. 10

HV / MUTANS STUDY NEWSLETTER

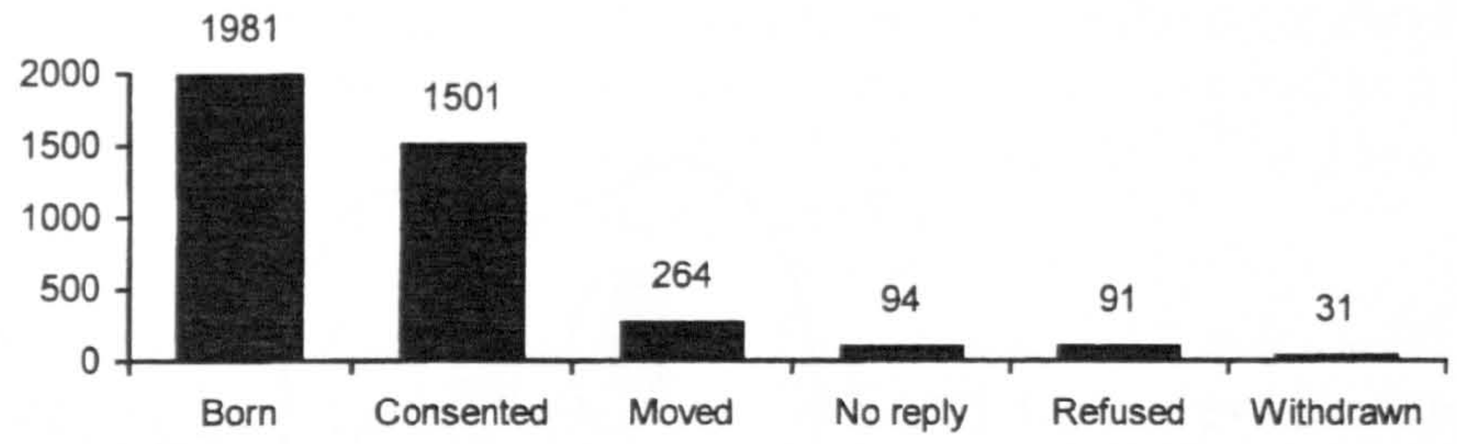
April 1998



Results

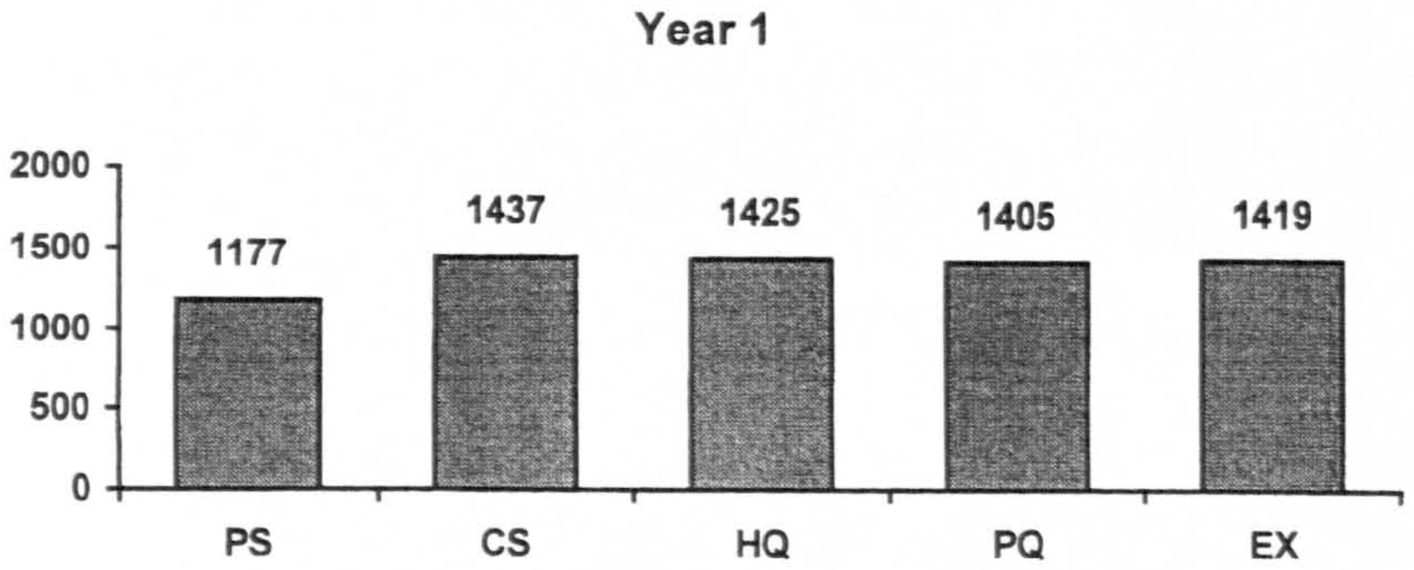
This newsletter is designed to provide you with some updated results of the study. They are from data collected for the children aged 1 to 3 years as we are still trying to collect the 4 year old data.

1. **Numbers of children born and consented:**



The diagram shows the numbers of children who were originally born and resident in Dundee between 1 April 1993 and 31 March 1994. We still hold consent forms for 1682 infants, although some of these families have now moved away from the area.

2. **Data collected for the infants at 1, 2 and 3 years of age:**



SHVA Conference - Pitlochry

Margaret Robertson and Heather will be speaking about the results of our study at the above conference on 8 May 1998.

It would be great to see as many of you there as possible. You can tell everyone how hard you have worked to make the study a success.

Again, a big thank you for your continued support.

HV/Mutans Study
Unit of Dental & Oral Health
1 Airlie Place
Dundee
DD1 4HQ

Tel: 01382 345755 / 345751

Sampling

As from Tuesday 2 May 1998 to 30 June 1998

Igloos will be at **Wallacetown Health Centre** only. These will be picked up at 16:30 daily. We appreciate that many of you are doing catch up clinics and extra sampling bags will be delivered upon request. Please just call Heather / Maureen on 345755/345751 to ask for sampling kits.

Remember if sample bags are used they must be returned on the same day as the bugs die after 8 hours. Unused bags may be kept in the fridge for 1 week.

4-year data

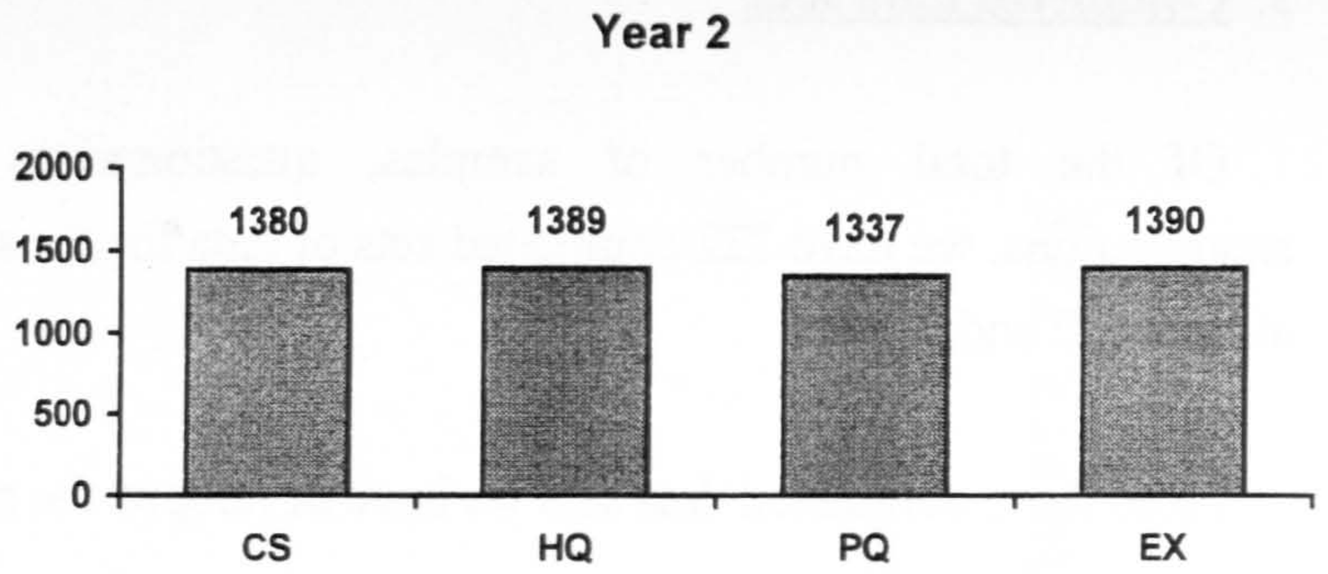
The end of this study is in sight (30 June for last samples and questionnaires) so we still have several weeks to collect 4-year data.

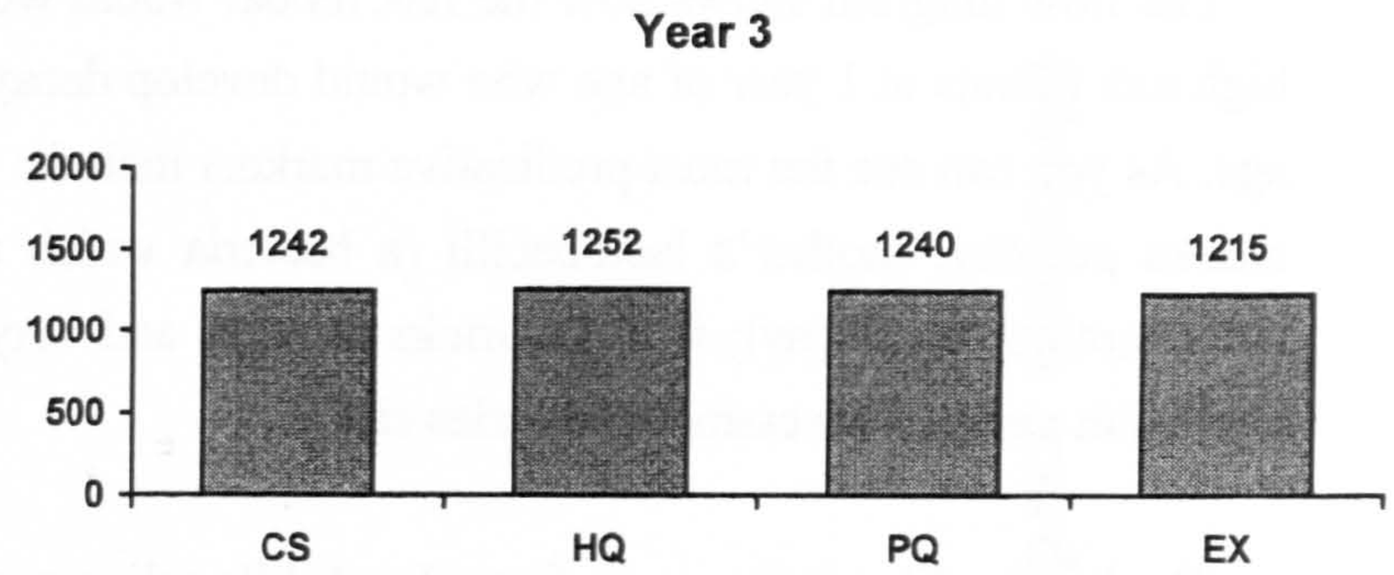
We desperately need more samples and questionnaires to enable a final risk model to be developed and to put Dundee health visitors on the map!

Feedback questionnaires

Enclosed is your final feedback questionnaire.

Please complete and return to Maureen as soon as possible as we always value your constructive opinions.





These graphs show the numbers of Parental saliva sample (PS), Child saliva samples (CS), Health Visitor and Parental questionnaires (HQ & PQ) and Dental Examinations (EX) carried out for the first 3 years.

3. **Completed data sets:**

Of the total number of samples, questionnaires and dental examinations, we have **722** completed sets of data for the **same** children at ages 1, 2 and 3 years.

From these completed data sets we have developed the risk model.

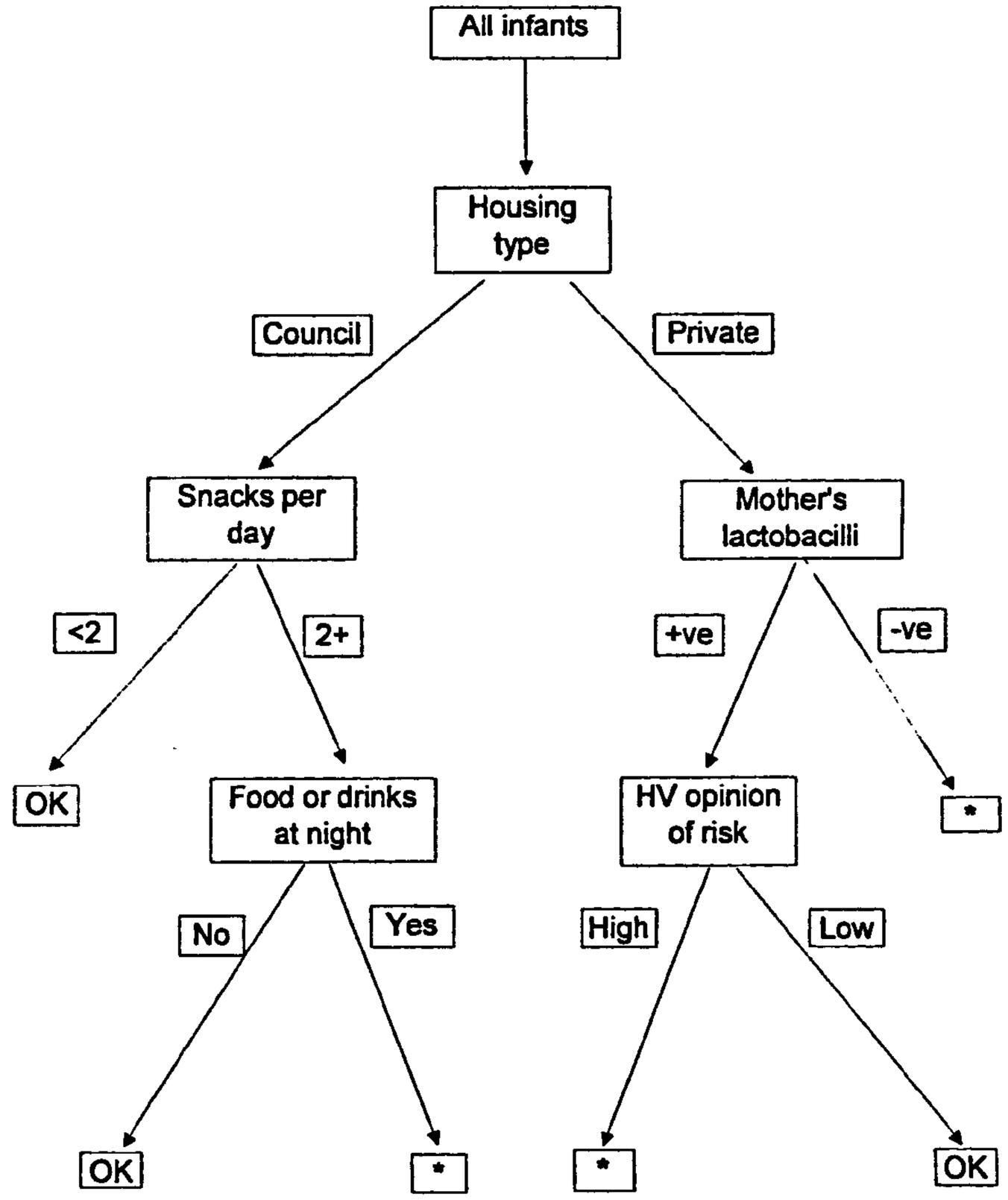
4. **The Dundee Caries-Risk Model (DCRM):**

The flow diagram shows how the risk model would work to identify high risk infants at 1 year of age who would develop decay at 3 years of age. As you can see the most predicative markers include: housing type; snacks per day, mother's lactobacilli (a bacteria which reflects sugar consumption and decay); food or drinks at night and very importantly, the health visitor's assessment of caries-risk.

The levels of bacteria grown from the child's saliva sample were not highly significant and considering the cost, cannot be regarded as an important risk indicator.

One main conclusion, therefore is that the most important predictors involve a few simple questions and are basically knowledge which health visitors already have of the family. More importantly, we can rely on your assessment of the child's risk status. Well done!

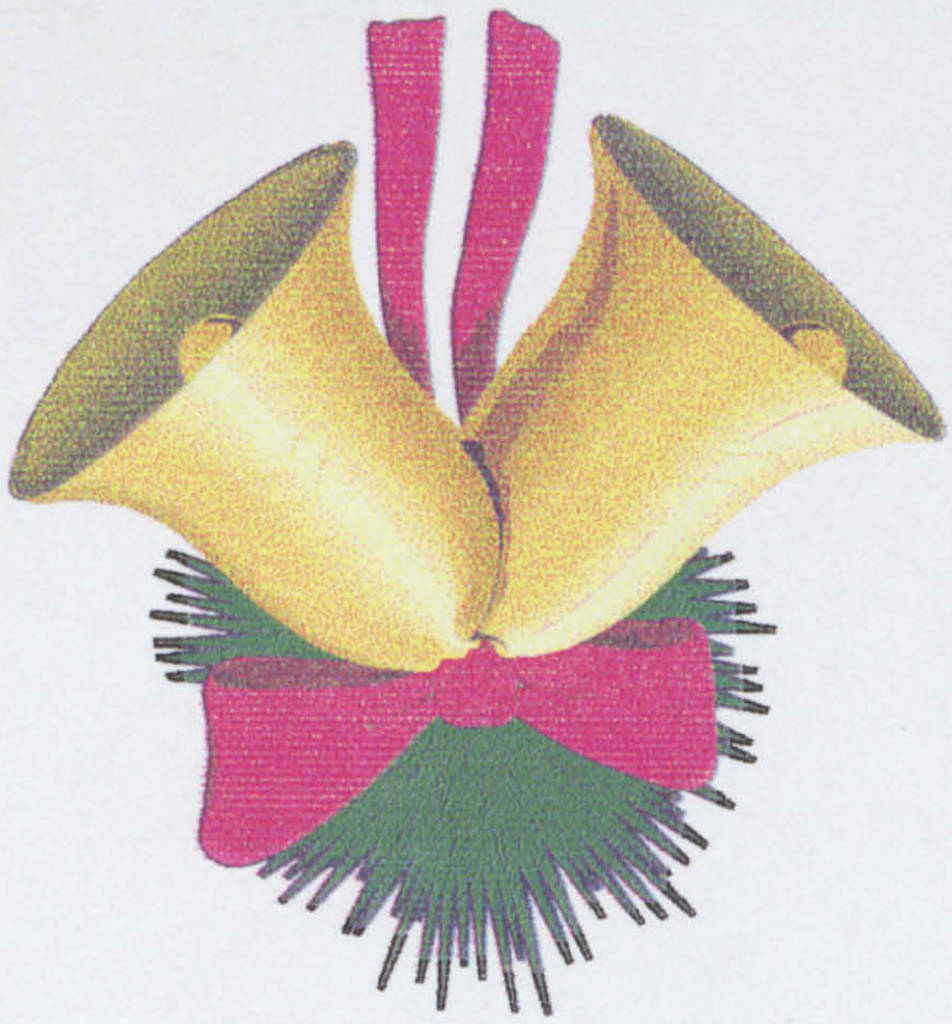
We will keep you updated on further analysis of data.



* target with preventive dentistry

3.12 Example of a Christmas card issued to health visitors

Printed by the Mutans Study Team



Seasons Greetings

Merry Christmas
and
A Happy New Year
from
The Mutans Study Team

4.1 Photograph of a dental examination in year-1 of study

Appendix 4.1 Photograph of a dental examination in year-1 of the study.

4.2 Dental examination data form

DENTAL EXAMINATION DATA

CHILD'S NAME | | **DATE OF EXAMINATION** | | | |

CHILD'S STUDY NUMBER | | **TYPE OF VISIT** | |

DATE OF BIRTH | | | | **SEX** **Male 1** **Female 2** | | **IS VISUAL PLAQUE PRESENT** **Yes 1** **No 2** | |

EROSION PRESENT AT 4 YEARS **Yes 1** **No 2** |

CLINICAL EXAMINATION

	Upper Right					Upper Left					Lower Right					Lower Left				
	e	d	c	b	a	a	b	c	d	e	e	d	c	b	a	a	b	c	d	e
M																				
O			X	X	X	X	X	X					X	X	X	X	X	X		
D																				
B																				
L																				

TOOTH CODES

Unerupted	**U**
Missing due to Caries	**6**
Missing due to Trauma	**T**
Excluded	**X**

SURFACE CODES

Present and Sound	**G**	Dentine lesion (non-C)	**D**	Filled, no decay	**F**
White spot lesion	**W**	Dentine Cavity	**C**	Filled and Decay	**5**
Brown spot lesion	**B**	Arrested dentine decay	**A**	Filled, needs Replacing (no decay)	**R**
Enamel cavity	**E**	Pulpal Involvement	**P**	Sealed surface	**S**

DENTAL EXAMINATION DATA

CHILD'S NAME | | **DATE OF EXAMINATION** | | | |

CHILD'S STUDY NUMBER |

DATE OF BIRTH | | | **SEX** **Male 1** **Female 2** |

FOTI EXAMINATION

	Upper Right					Upper Left					Lower Right					Lower Left				
	e	d	c	b	a	a	b	c	d	e	e	d	c	b	a	a	b	c	d	e
M																				
O			X	X	X	X	X	X					X	X	X	X	X	X		
D																				
B																				
L																				

FOTI CODES

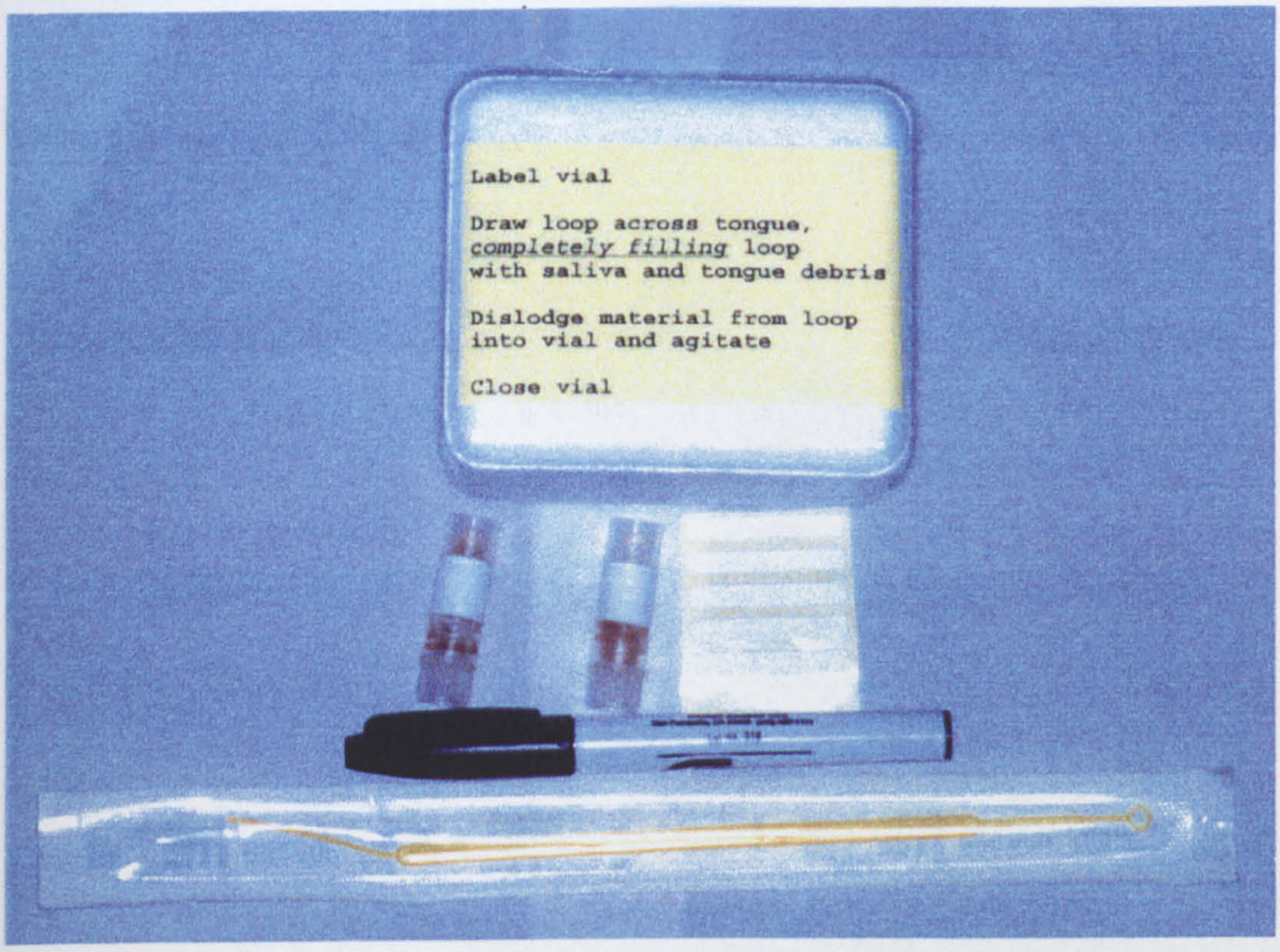
Shadowing in Enamel **E**
Shadowing in Dentine **D**

Dentists Assessment - Is child at High-caries risk? **Yes 1** **No 2** |

4.3 Photograph of styrofoam storage box contained within saliva sampling kit

Appendix 4.3 Photograph of styrofoam storage box contained within sampling kit.

4.4 Photograph of contents of saliva sampling kit



Appendix 4.4 Photograph of contents of saliva sampling kit.

4.5 Photograph of 'fresh' and 'used' igloos

Appendix 4.5 Photographs of *fresh* and *used* igloos

4.6 Photograph of random page from microbiological logbook

Study No	Name	Date	Sample No
2205930117		24/9	5896
1608930165		"	5897
2109930228		"	5898
2312930218		"	5899
1508930317		"	5900
1010930117		"	5901
0809930390		"	5902
1908930179		"	5903
0301940150		"	5904
2408930332		"	5905
2208930282		"	5906
	THURS		
2408930154		25/9	5907
0311930387		"	5908
1909930288		"	5909
2609930445		"	5910
0308930150		"	5911

4.7 Instructions for catalase test

Catalase Test

This test is based on the principle that the enzyme catalase breaks down hydrogen peroxide (H_2O_2), resulting in the formation of bubbles.

1. Carefully pick off a culture from the agar plate and place onto a microscope slide
2. Pipette one drop of hydrogen peroxide onto the colony
3. Observe presence / absence of instant bubbling

Interpretation

Presence of bubbling	Catalase +ve
Absence of bubbling	Catalase -ve

4.8 Instructions for Gram's stain

Gram's stain

1. Preparation of a heat fixed slide

Use a sterile loop

Spread loopfull of material on slide: keep clear of edges

Dry film in air or by holding high over bunsen flame

Fix film on slide by slowly passing it three times through bunsen flame

Allow to cool before staining

2. Gram's staining method

a) Flood the slide with methyl – violet solution, allow to act for 5 minutes

b) Wash off stain with iodine solution

c) Allow iodine to act for 2 minutes

d) Drain off excess iodine. Decolourise with acetone for not more than 5 seconds

e) Wash slide immediately in water

f) Apply basic fuchsin counter stain for 30 seconds

g) Wash in water, blot and air dry

Interpretation of Gram's staining method

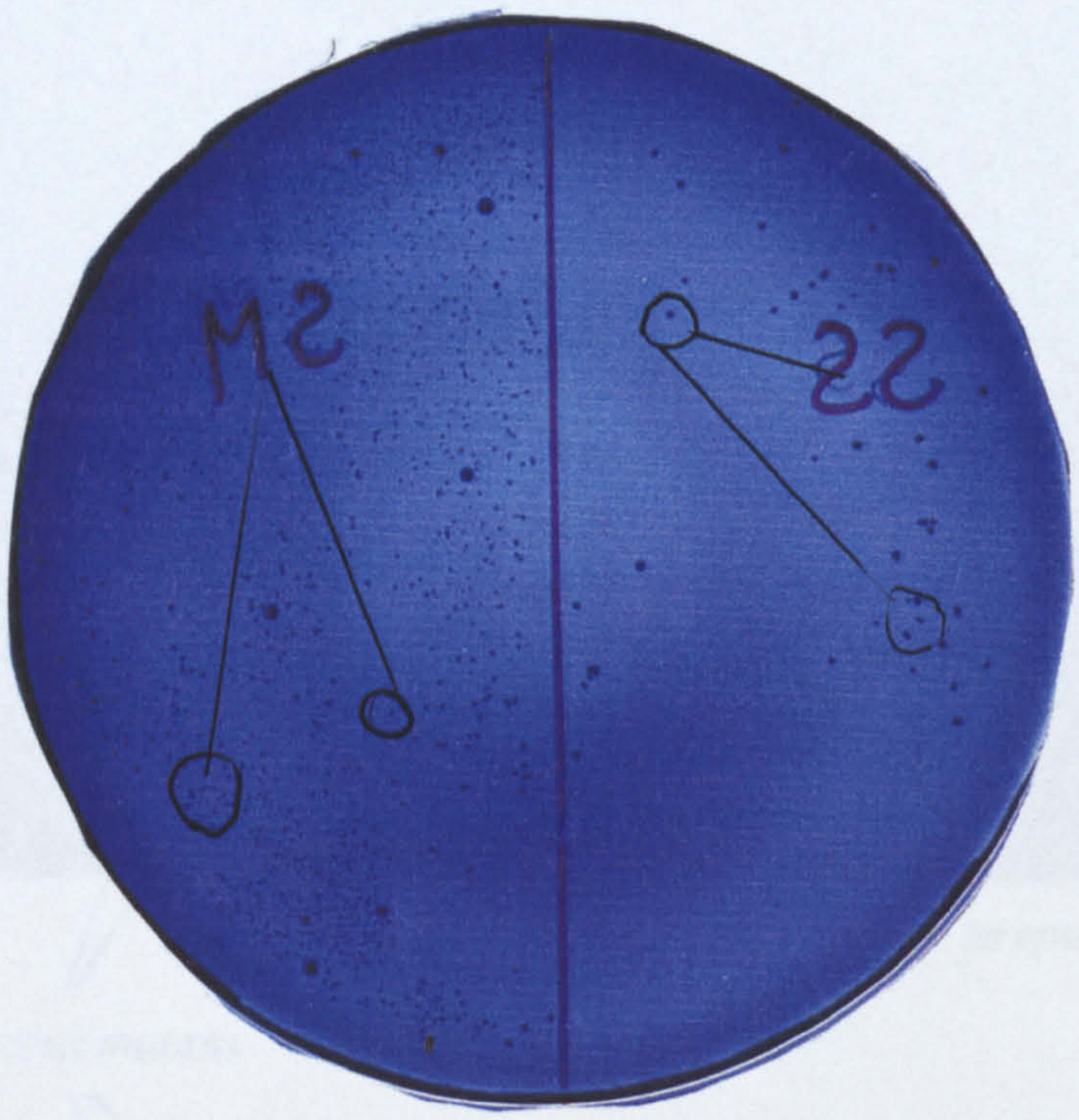
1. Before acetone decolourisation all organisms appear Gram +ve
2. After acetone decolourisation those organisms which are Gram +ve are no longer visible
3. These Gram –ve organisms are visualised after the application of the counterstain

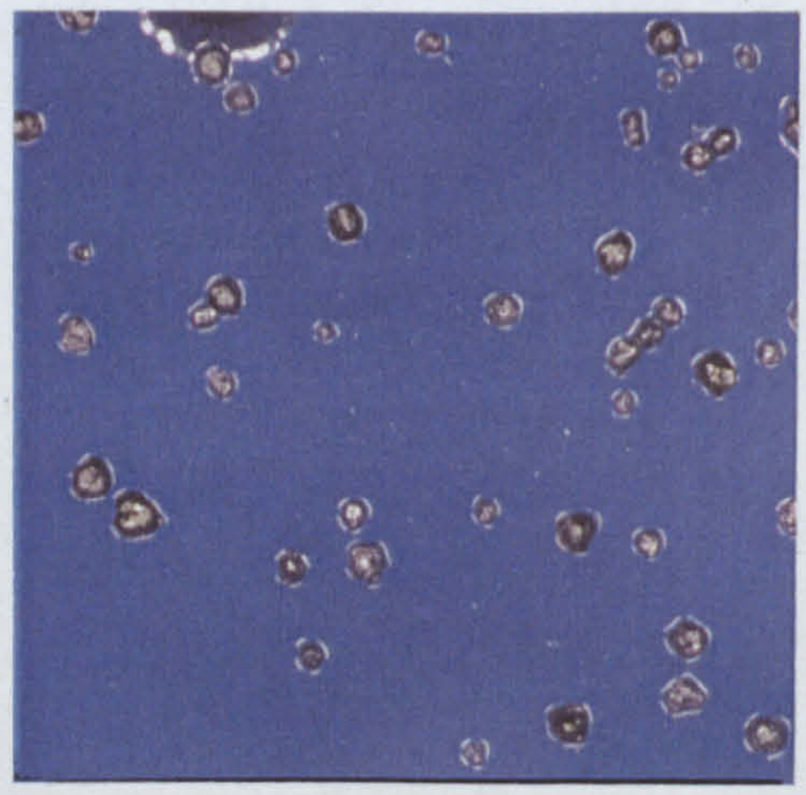
The division occurs due to cell wall. In G+, the cell wall shrinks in the presence of acetone, trapping the crystal violet / iodine.

4.9 Microbiological data sheet

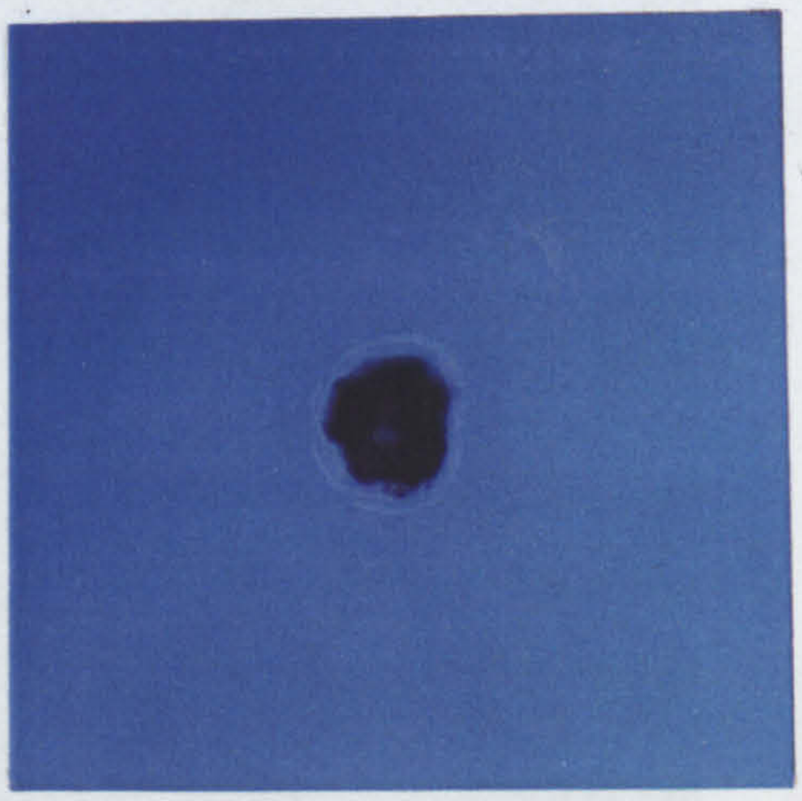
1 [ ] 2 [ ]

	CHILD NUMBER	SLIDE NO.	MEDIA	NO. OF C.F.U's	DESCRIPTION OF COLONIES	CAT TEST	PRES DIAG	DEF DIAG	L/D
1									
	CHILD NUMBER	SLIDE NO.	MEDIA	NO. OF C.F.U's	DESCRIPTION OF COLONIES	CAT TEST	PRES DIAG	DEF DIAG	L/D
2									



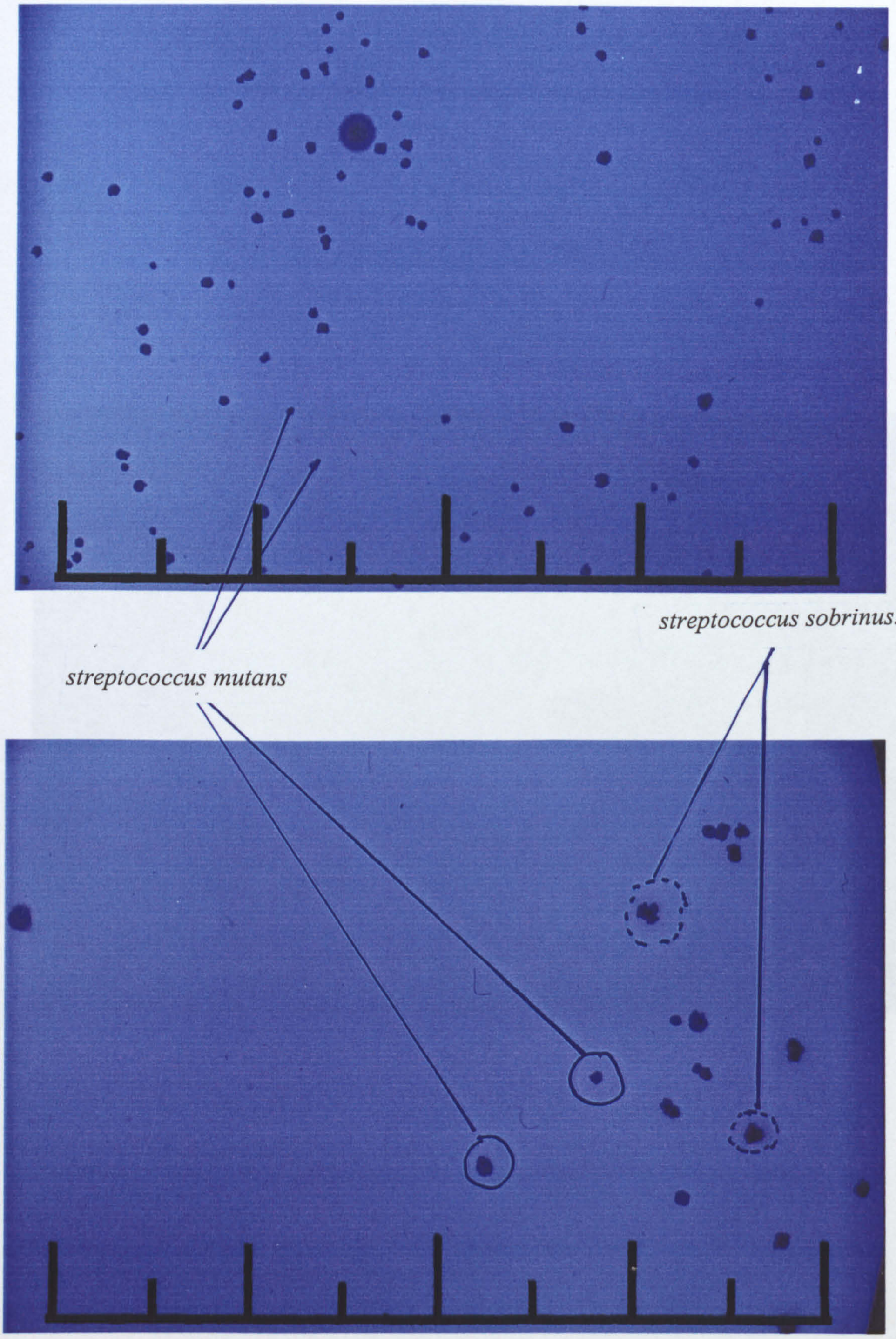


streptococcus mutans



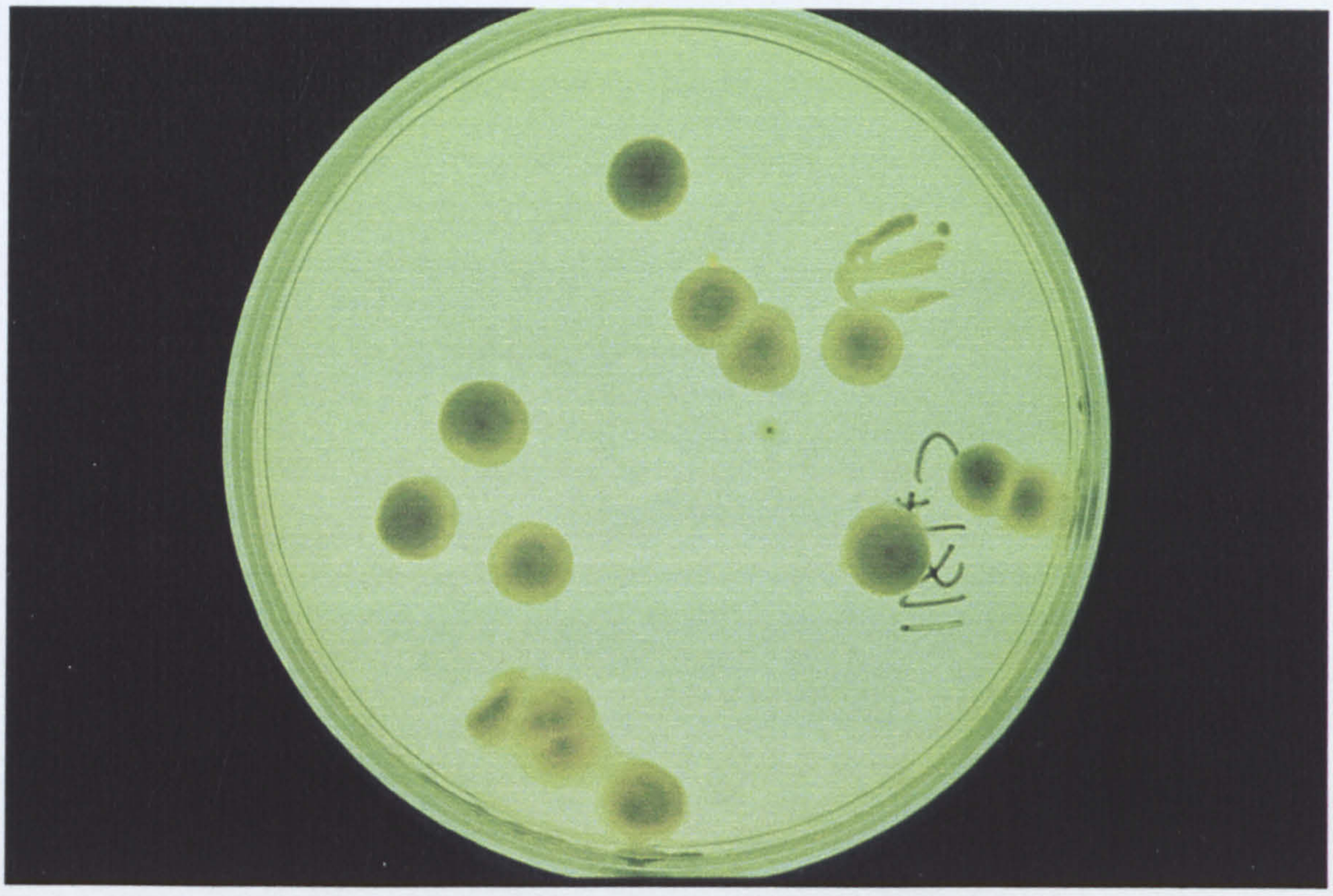
streptococcus sobrinus

Appendix 4.10 Photograph of BMSA plate with colonies of *streptococcus mutans* and *streptococcus sobrinus*.



Appendix 4.10 Photograph of BMSA plate with colonies of *streptococcus mutans* and *streptococcus sobrinus*.

Appendix 4.11 Photograph of a Rogosa plate with colonies of lactobacillus species.



Appendix 4.12 Photograph of Sabouraud dextrose agar with colonies of yeast species

4.13 Charts of enzyme substrate tests carried out for confirmatory identification of caries associated microorganisms in London

SAMPLES FROM DUNDEE.

3T x 8
~280

PLATE NO. 1a **DATE** 18.9.96 **INITIALS** RL

STRAIN NO.	AMY	ARB	IN	LAC	MAN	MEL	NAG	RAF	SOR	AES	ARG	PUR
1930B					±	+	−		−	+	+	
1240B					+	+	+		−	−	+	
1409B					−	−	+		−	−	−	
1262B					+	+	+		+	+	−	
12135B					−	−	−		−	−	+	
1303B					+	+	+		+	+	−	
1323B					−	+	−		−	−	+	
1390B					−	−	−		−	−	+	

PLATE NO. 1b **DATE** 18.9.96 **INITIALS** RL

STRAIN NO.	AMY	ARB	IN	LAC	MAN	MEL	NAG	RAF	SOR	AES	ARG	PUR
1253B					−	−	−		−	−	−	
1255B ~~1211B~~					−	+	−		−	+	+	
1211B ~~1346B~~					+	+	+		+	+	+	
1346B ~~1255B~~					+	+	+		+	+	+	
1278B					+	+	+		+	+	−	
1315B					+	+	+		+	+	−	
1416B					−	+	−		−	+	+	
1347B					+	+	+		+	+	−	

PLATE NO. 2a **DATE** 18.9.96 **INITIALS** RL

STRAIN NO.	AMY	ARB	IN	LAC	MAN	MEL	NAG	RAF	SOR	AES	ARG	PUR
1324B					−	−	+		+	−	+	
1361Bn.					+	+	+		+	?	−	
1898B					+	−	+		+	?	−	
1949B					−	−	−		−	−	−	
1388B					−	+	−		−	+	+	
1898B					−	+	+		−	−	+	
1320B					−	−	+		−	+	+	
1964B					+	+	+		+	+	−	

PLATE NO. 2b **DATE** 18.9.96 **INITIALS** RL

STRAIN NO.	AMY	ARB	IN	LAC	MAN	MEL	NAG	RAF	SOR	AES	ARG	PUR
1946B$_2$					−	+	+		+	+	+	
[redacted]												
1949B					+	+	+		+	+	−	
2021B					+	±	+		+	+	−	
1963B					+	+	+		+	+	−	
1980B					+	−	+		+	−	−	
1976B					+	+	+		+	+	+	
1982B					+	−	+		+	−	+	

Plate	No. subbed	S.mutans	No Growth	Not mutans
4823B	2	1	1	
504	8	3	5	
4824B	6	6		
1T	6	6		
B	4	4		
C2	3	-	2	1
2560	7	7	2	
4820B	9	9		
4800B	8	8		
1402B	8	8		
4698B	11	10	1	
2T	8	7	1	
AF3	10	-	5	5
4821B	10	5	5	

All isolates identified as S.mutans produced acid from N-acetylglucosamine, mannitol, sorbitol and melibiose and arginine negative and were usually aesculin positive.

The reasons for the no-growth subcultures is probably due to the age of the paltes. We would normally subculture on the day of removal from the incubator.

Of the 79 strains which did grow 73 were S.mutans. This represents an overall success rate of 92 percent.

I suggest that this exercise be repeated every 3-4 months.

22/11/96.

4.14 Training of study technician and study dentist in microbiological methodology

TRAINING OF C.R.F. AND TECHNICIAN

DATE: 14th - 16th February 1994

Location: Oral Microbiology Laboratory,
R.C.S. Department of Dental Sciences,
King's College,
School of Medicine and Dentistry,
Caldecot Road,
London. SE5 9RW.
Tel: 071-326-3608 ext. 2586

Supervisor: Dr. D. Beighton.

Aim of visit

To extend knowledge and understanding of techniques required in oral microbiological sampling and processing, with specific relation to the Health Visitor/Mutans research study.

1. Preparation of Media.

Mitis-salivarius agar with bacitracin (BMSA)

As we had no prior experience of preparing or using this agar, this information proved most useful. We were able to observe the correct way of handling the equipment, and, fortunately realise

the cycle of our autoclave required a reduction in time to prevent denaturisation of the nutrients in the agar.

Sabouraud agar (SAB)

Again the only modification of our techniques required was alteration of the autoclave cycle. The plates we had taken to London with us did not have the correct growth of yeasts, and the agar itself was a different colour to that being produced in this Laboratory. We had to reduce the time of sterilisation to avoid damage to the agar.

Rogosa agar (ROG)

The technique used for preparation of this media were identical to our own. This media does not need to be autoclaved.

2. Plating techniques.

The plating techniques demonstrated to us were identical to those being executed in Dundee. Isolation of single colonies with platinum loops and wire were observed. This involved spreading from a sample well on the plate, followed by a final streak into the centre of the plate, where single colonies would then be found. This procedure may be used when colonies are required to be regrown and stored on plates for an extended period of time. This procedure can be repeated as often as required. The result being the growth of isolated colonies of the same species.

3. Identification of colonies.

The appearance, odour, and structure of colonies of bacterial and fungal species on selective media were demonstrated. Each species have their own individual features, and are identified thus.

a) M.S.A. with BACITRACIN.

This agar itself was blue in colour and recognisable from this feature alone. There were numerous colony types on each plate. The samples were taken from the saliva of the researchers in the Laboratory. Colonies of Streptococcus mutans and Streptococcus sobrinus were identified. These both had individual characteristics as follows.

Steptococcus mutans.

Dark blue colour

Crenated edges

Raised above media

Raspberry shaped

Incorporated into media

Very difficult to remove

Streptococcus sobrinus.

The characteristics were identical to above and additionally were surrounded by a halo.

Such an approach of differentiating these species, however, could severely underestimate the number of S. sobrinus colonies, and it is imperative that bacterial colonies presumed to be different on the basis of their morphology should be adequately characterised by biochemical or serological techniques. (de Soet *et al*, 1987) reference still to be photocopied!.

b) ROGOSA AGAR.

The rogosa media was identical in all respects to that being used in Dundee. The colonies were medium in size, white and creamy, some were larger or smaller and varied slightly. After Gram Staining, it was thought that all of these were Lactobacilli species. It was therefore decided that the techniques being used in Dundee were acceptable.

c) SABOURAUD AGAR.

As previously mentioned, this media had been autoclaved for far longer than was required in Dundee. It was observed that the colonies grown were indeed much different to those we had plated. Another major difference was in the odour. In London the colonies had a very distinctive 'brewer's yeast' odour. This is a strong indicator of the presence of Candidal species. We had not been able to grow this, probably due to the changes the media had undergone during autoclaving. These colonies also had a very distinctive appearance being large, white and very creamy. On gram staining of these species a positive result for yeast was obtained.

However, again, it must be noted that for definitive identification further tests were required.

4. Serological and Biochemical Techniques.

The procedures used in the Laboratory for definitive identification were demonstrated. There were no facilities in the laboratory in Dundee for these tests, but they were essential for back up diagnosis and also as a double check that the correct organisms were indeed being grown. Frozen samples and plates sent regularly from Dundee will undergo these further tests.

a) Sugar fermentation tests.

A copy of this procedure is attached. The colonies were incubated in Todd Hewitt broth for 48hours. 135ul of sugar was added to sterile microlitre trays (Corning cell Wells) with a control row at the end. On addition of 45ul of incubated broth, the trays were incubated anaerobically at 37oC for 24 hours. Yellow indicated a positive result for the well, purple being negative. For arginine hydrolysis, 45ul of Nesslers reagent was added to the well. Orange colour indicated hydrolysis and a positive result. All these results were noted in chart like form and stored.

b) Enzyme substrate tests.

Non - sterile microlitre trays (Medicell) were used for this test. Organisms were removed directly from the plate with a sterile cotton swab. These were suspended in approximately 1ml of TES buffer. 20 ul of enzyme was placed into each well followed by 45ml of each isolate then incubated at 37oC for 3 hours. Substrate hydrolysis was determined by measuring fluorescence on the Perkin Elmer fluorimeter. Production of enzyme hydrolysis was positive if an increase in fluorescence of 5 units above the control standard was present. This was viewed on the U.V. light box.

5. Gram staining

The reagents and methodology used in London were identical to those of Dundee. After provisional identification of colonies on the plates, these were gram stained, and the results checked by Dr. Beighton to ensure we were achieving a satisfactory result.

6. Transfer of samples.

It was decided by Dr. Beighton that initially all plates onto which samples had been cultured would be transported to London in styrofoam storage boxes. Prior to sending these we would remove a colony, making a provisional diagnosis from colony characteristics, gram staining and catalase testing. These colonies would then undergo identical testing and also the additional tests discussed earlier. This then acts as a double check on all the techniques being used in Dundee. After a period of time, during which diagnostic consistency is achieved, a lesser number of colonies will be sent, and these will be in the frozen form in the protect vials, on average once per month. The technique for placing samples into the protect vials was demonstrated. A single colony was removed either from a plate or from broth,(however for our purposes, only the plates were applicable) and following agitation with the loop, the vial was inverted gently 16 times on average. The excess liquid from the vial was then siphoned using a sterile, plastic pasteur pipette. This transport medium was present in the vial on purchase. The

vial was then placed into a -80oC frezzer and stored as long as necessary.

Plating from the protect vials was also demonstrated. One bead was removed from the vial very carefully with a sterile loop. A well was made with the bead on the plate and streaked in the usual manner. A different bead was used for each different plate type, and the plates then incubated for 3 days. Further plating could then be carried out to maintain colony viability if required.

The protect vials transported to London with us were grown in this way onto Columbia, rogosa and RSBA. We then gram stained these, and Dr. Beighton checked our results. It was found that the sample contained Lactobacilli which was consistent with our findings in Dundee.

In conclusion, the visit to the Laboratory in London was most benificial to our training in microbiological techniques.

4.15 Microbiological Standard Operating Procedures (SOP) Booklet

Health Visitor / Mutans Study
University of Dundee

Standard Operating Procedures

Distribution

Professor N B Pitts

Dr. J Radford

Mrs. V Wilson

Miss H Ballantyne

Contents

1. Hazards

Care should be taken to avoid the inhalation of powdered agar. The risk is sufficiently low to negate the need for wearing protective gloves and masks, or working in a designated risk area.

Autoclaves should be used according to local rules

Bacitracin as an antibiotic has to be handled in such a way as to minimise the operator's exposure.

Gram's stain chemicals should be handled in such a way as to minimise the operator's exposure

Any use of naked flames for surface sterilisation should be accompanied with care to avoid personal injury and only after removal of inflammable materials from adjacent areas.

All media plates should be poured using an aseptic technique inside a laminar flow cabinet.

Glass ampoules should be scored with a diamond pencil and snapped open inside a paper tissue to minimise the risk of skin laceration.

Virkon should be handled with minimal contact and care and prolonged exposure avoided.

U.V. light on laminar flow cabinet should only be on when laboratory empty, or when eye protection is worn by operators.

2. Preparation of Media

2.1 BMSA Agar

Mitis salivarius agar is made up in accordance with the manufacturer's instructions.

87g of agar powder is suspended in 1 litre of distilled water

150g per litre of sucrose is added

medium is then brought to boil in autoclave at 121°C for 15 minutes

After autoclaving medium is cooled to 55°C and 1ml of 1% filter sterilised -potassium tellurite solution and 10ml (200units) of filter sterilised bacitracin are added per litre of medium.

The potassium tellurite solution is made from 2ml stock solution bought in glass ampoules. These are opened as detailed in 'hazards.' The solution is made up to 7ml by adding 5ml of de-ionised water. The final solution is filtered through a sterile 0.2µl cellulose acetate filter directly into a sterile vessel. This solution is then stored at < 8°C until required.

The bacitracin stock solution is prepared by dissolving 2000 units of bacitracin in 100ml of deionised water and sterilised as for potassium tellurite.

During the mixing of the medium after the addition of the bacitracin and potassium tellurite, care must be taken to avoid the formation of air bubbles. The media is poured onto the petri --dishes in a laminar flow cabinet following an aseptic technique.

The plates are labelled, wrapped and stored at < 8°C until required

Plates should only be used if the agar is less than one week old.

2.2 Rogosa agar

Rogosa agar should be made up according to manufacturer's instructions

suspend 82g of powder in 1 litre of distilled water

bring to the boil to dissolve completely

add 1.32ml of glacial acetic acid and mix thoroughly

heat to 90 - 100°C for 2-3 minutes with frequent agitation

place in water bath until cool enough to pour

pour in laminar flow cabinet using an aseptic technique

place in drying cabinet for 20 minutes

pack & store as BMSA agar.

CAUTION: Care must be exercised when handling glacial acetic acid.

2.3 Sabouraud Dextrose Agar

SAB agar is made up according to manufacturer's instructions

suspend 65g of powder in 1 litre distilled water

bring to the boil to dissolve completely

sterilise by autoclaving at 121°C for 15 minutes

after autoclaving place in water bath (using fresh distilled water) at 50°C

allow to cool

pour plates in laminar flow cabinet when cool enough to avoid large amounts of condensation

dry, wrap and store as for other agar.

3. Preparation of sampling kits

3.1 Preparation of Fastidious Anaerobic Broth

Prepare according to manufacturer's instructions

disperse 29.7g of powder in 1 litre of distilled water

soak for 10 minutes

bring to the boil with gentle mixing

autoclave at 121°C for 15 minutes

dispense into sterile 1ml vials using sterile pipette in laminar flow cabinet

3.2 Preparation of cool - bags

Daily: 13 cool - bags containing each — 1 sterile loop

set of labels

pentel pen

Poly box with 1 fresh vial

Igloo 1 — 8 freezer packs + 7 cool - bags

Igloo 2 — 8 freezer packs + 9 cool - bags

Also prepare 1 cool - bag with 20 loops and 20 vials for Heather.

Prepare igloos ready for sample pick - ups at 1600 hours.

Fresh vials with FAB made up weekly

Cool - bags returned from Health Centres re - used, and all vials discarded weekly.

4. Sample identification, plating out and incubation

Samples returned to Laboratory at approximately 1600 hours

Sample number and study number of infant entered into log book

Plated numbered with sample number and letter to indicate media (R - rogosa), (B - BMSA), (S - Sabouraud)

Each sample vial is mixed by vortexing for 10 seconds

100micro litres from each vial is pipetted onto each plate

Plates are spread one at a time using a disposable L - shaped spreader

Once all samples have been plated out, BMSA and rogosa are taped together, and SAB plates are taped separately.

SAB plates are labelled with day to be removed from incubator, then placed directly into incubator.

ROG and BMSA plates are also labelled with removal date then placed in an anaerobic jar

The anaerobic gas pack is opened and after addition of 10mls of distilled water, quickly placed into the jar, and the jar closed as soon as possible to prevent CO_2 loss.

The anaerobic jar is labelled with the date for removal.

All plates are incubated for 72 hours at 37°C.

5. Presumptive identification and quantification of bacteria and yeasts

On removal from incubator all plates are kept refrigerated overnight to improve visual appearance of individual colonies.

5.1 BMSA agar

'Protect Vial' one representative colony of mutans streptococci form each plate

If over 100 colonies, 'Protect Vial' 2 colonies

Gram Stain a representative of each colony morphology from every 100th plate

5.2 Rogosa agar

Gram Stain a representative of each colony morphology.

'Protect Vial' a representative of each colony morphology every 10th plate

5.3 Sabouraud Dextrose agar

Gram Stain a representative of each colony morphology which is Catalase +

5.4 Identification sheets

Must be completed for each sample.

Box labelled 'PRES DIAG' must be filled in.

6. Storage and transportation of colonies

6.1 'Protect Vials'

Pick off colony to be sent for definitive identification with sterile loop provided.

Place into labelled vial. replace cap, and invert vial 16 times.

Remove cap and withdraw as much liquid as possible with disposable sterile pipette.

Replace cap and store vial at -70°C, having fast frozen first in domestic freezer.

6.2 Agar plates

Tape plates together in stacks

Wrap each stack of plates in plastic bubble sheets

Place stacks in box lined with plastic bubble wrap.

Firmly tape box and wrap whole package with plastic bubble wrap

Label the box clearly, and dispatch to Dr. Beighton's laboratory as soon as possible by Courier.

5.1 Cross-tabulations for caries diagnosis reproducibility

HBD1A * HBD1B * YY Crosstabulation

Count

YY			HBD1B		Total
			d	x	
94	HBD1A	d	50	4	54
		x	1	5	6
	Total		51	9	60
96	HBD1A	d	41	2	43
		x	3	14	17
	Total		44	16	60
97	HBD1A	d	43	1	44
		x		16	16
	Total		43	17	60

Symmetric Measures

YY			Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
94	Measure of Agreement	Kappa	.621	.153	4.941	.000
	N of Valid Cases		60			
96	Measure of Agreement	Kappa	.791	.089	6.133	.000
	N of Valid Cases		60			
97	Measure of Agreement	Kappa	.958	.041	7.429	.000
	N of Valid Cases		60			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

HBD3A * HBD3B * YY Crosstabulation

Count

YY			HBD3B		Total
			d	x	
94	HBD3A	d	37	1	38
		x	2	20	22
	Total		39	21	60
96	HBD3A	d	38	1	39
		x		21	21
	Total		38	22	60
97	HBD3A	d	37		37
		x		23	23
	Total		37	23	60

Symmetric Measures

YY			Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
94	Measure of Agreement	Kappa	.891	.061	6.909	.000
	N of Valid Cases		60			
96	Measure of Agreement	Kappa	.964	.036	7.470	.000
	N of Valid Cases		60			
97	Measure of Agreement	Kappa	1.000	.000	7.746	.000
	N of Valid Cases		60			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

25.2.08

JPD1A * JPD1B * YY Crosstabulation

Count

YY			JPD1B d	JPD1B x	Total
97	JPD1A	d	38	6	44
		f	2		2
		x		14	14
	Total		40	20	60

K = 0.692

Symmetric Measures

YY			Value
97	Measure of Agreement	Kappa	.[a]
	N of Valid Cases		60

a. Kappa statistics cannot be computed.They require a symmetric 2-way table in which the values of the first variable match the values of the second variable.

JPD3A * JPD3B * YY Crosstabulation

Count

YY			JPD3B d	JPD3B f	JPD3B x	Total
97	JPD3A	d	29		1	30
		f	2	1		3
		x	3		24	27
	Total		34	1	25	60

Symmetric Measures

YY			Value _	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
97	Measure of Agreement	Kappa	.811	.071	6.928	.000
	N of Valid Cases		60			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

HBD1A * CLD1A * YY Crosstabulation

Count

YY			CLD1A d	CLD1A x	Total
96	HBD1A	d	42	1	43
		x	1	16	17
	Total		43	17	60

Symmetric Measures

YY			Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
96	Measure of Agreement	Kappa	.918	.057	7.110	.000
	N of Valid Cases		60			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

HBD3A * CLD3A * YY Crosstabulation

Count

YY			CLD3A		Total
			d	x	
96	HBD3A	d	39		39
		x		21	21
	Total		39	21	60

Symmetric Measures

YY			Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
96	Measure of Agreement	Kappa	1.000	.000	7.746	.000
	N of Valid Cases		60			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

HBD1A * JPD1A * YY Crosstabulation

Count

YY			JPD1A			Total
			d	f	x	
97	HBD1A	d	40	2	2	44
		x	4		12	16
	Total		44	2	14	60

Symmetric Measures

YY			Value
97	Measure of Agreement	Kappa	.[a]
	N of Valid Cases		60

a. Kappa statistics cannot be computed.They require a symmetric 2-way table in which the values of the first variable match the values of the second variable.

HBD3A * JPD3A * YY Crosstabulation

Count

YY			JPD3A			Total
			d	f	x	
97	HBD3A	d	29	3	5	37
		x	1		22	23
	Total		30	3	27	60

Symmetric Measures

YY			Value
97	Measure of Agreement	Kappa	.[a]
	N of Valid Cases		60

a. Kappa statistics cannot be computed.They require a symmetric 2-way table in which the values of the first variable match the values of the second variable.

Crosstab

Count

		JPD1MF			Total
		d	f	x	
HBD1MF	d	43	2	9	54
	x	1		5	6
Total		44	2	14	60

Kappa = 0.368

Crosstab

Count

		CLD1MF		Total
		d	x	
HBD1MF	d	42	12	54
	x	1	5	6
Total		43	17	60

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Measure of Agreement	Kappa	.337	.129	3.151	.002
N of Valid Cases		60			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

Crosstab

Count

		CLD1MF		Total
		d	x	
JPD1MF	d	40	4	44
	f	2		2
	x	1	13	14
Total		43	17	60

Kappa = 0.714

Crosstab

Count

		JPD3MF			Total
		d	f	x	
HBD3MF	d	28	3	7	38
	x	2		20	22
Total		30	3	27	60

Kappa = 0.614

Crosstab

Count

		CLD3MF		Total
		d	x	
HBD3MF	d	37	1	38
	x	2	20	22
Total		39	21	60

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Measure of Agreement	Kappa	.891	.061	6.909	.000
N of Valid Cases		60			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

Crosstab

Count

		CLD3MF		Total
		d	x	
JPD3MF	d	29	1	30
	f	3		3
	x	7	20	27
Total		39	21	60

Kappa = 0.646

original .dbf
HB intra-examiner
Kappa.

IN VIVO
57 CHILDREN. 1140 TEETH

D1MFTA * D1MFTB Crosstabulation

Count

		D1MFTB			
		d	m	x	Total
D1MFTA	d	69	2	25	96
	m		4		4
	x	27	2	1011	1040
Total		96	8	1036	1140

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Measure of Agreement	Kappa	.700	.037	24.791	.000
N of Valid Cases		1140			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

D3MFTA * D3MFTB Crosstabulation

Count

		D3MFTB			
		d	m	x	Total
D3MFTA	d	15	2	4	21
	m		4		4
	x	11	2	1102	1115
Total		26	8	1106	1140

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Measure of Agreement	Kappa	.671	.069	26.383	.000
N of Valid Cases		1140			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

IN VIVO HM x JP.
$d_1 + d_3$.

Crosstab

Count

		JPD1MF			Total
		d	f	x	
HBD1MF	d	10			10
	f		3	1	4
	x	6	1	179	186
Total		16	4	180	200

d1mf

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Measure of Agreement	Kappa	.748	.085	12.823	.000
N of Valid Cases		200			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

Crosstab

Count

		JPD3MF			Total
		d	f	x	
HBD3MF	d	1		1	2
	f		3	1	4
	x	1	1	192	194
Total		2	4	194	200

d3mf

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Measure of Agreement	Kappa	.659	.160	11.458	.000
N of Valid Cases		200			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

teeth. dbf in vivo 10 children.

5.2 Cross-tabulations for identification of caries associated microorganisms reproducibility

Val vs HB.

SMV * SMH

Crosstab

Count

		SMH		Total
		.00	1.00	
SMV	.00	1		1
	1.00		19	19
Total		1	19	20

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Measure of Agreement	Kappa	1.000	.000	4.472	.000
N of Valid Cases		20			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

LACTV * LACTH

Crosstab

Count

		LACTH		Total
		.00	1.00	
LACTV	.00	4		4
	1.00		16	16
Total		4	16	20

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Measure of Agreement	Kappa	1.000	.000	4.472	.000
N of Valid Cases		20			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

YEASTV * YEASTH

Crosstab

Count

		YEASTH		Total
		.00	1.00	
YEASTV	.00	10		10
	1.00		10	10
Total		10	10	20

STREP CROSS -

- TABULATIONS.

- VAL- VAL·OBF

VAR00001 * VAR00002 Crosstabulation

Count

		VAR00002		
		.00	1.00	Total
VAR00001	.00	16		16
	1.00		24	24
Total		16	24	40

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Measure of Agreement	Kappa	1.000	.000	6.325	.000
N of Valid Cases		40			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

val_val.dbf

Crosstabs

LACTA * LACTB

Crosstab

Count

		LACTB		Total
		.00	1.00	
LACTA	.00	11		11
	1.00		19	19
Total		11	19	30

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Measure of Agreement	Kappa	1.000	.000	5.477	.000
N of Valid Cases		30			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

YEASTA * YEASTB

Crosstab

Count

		YEASTB		Total
		.00	1.00	
YEASTA	.00	14		14
	1.00	2	14	16
Total		16	14	30

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Measure of Agreement	Kappa	.867	.090	4.793	.000
N of Valid Cases		30			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Measure of Agreement	Kappa	1.000	.000	4.472	.000
N of Valid Cases		20			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

5.3 Correlation matrix data analysis

Correlations

		DEPCAT	SMM	SSM	STM	LACTM
DEPCAT	Pearson Correlation	1.000	.055	-.002	.060	.085**
	Sig. (2-tailed)	.	.088	.961	.065	.008
	N	1364	950	950	950	974
SMM	Pearson Correlation	.055	1.000	.172**	.969**	.196**
	Sig. (2-tailed)	.088	.	.000	.000	.000
	N	950	951	951	951	951
SSM	Pearson Correlation	-.002	.172**	1.000	.264**	.091**
	Sig. (2-tailed)	.961	.000	.	.000	.005
	N	950	951	951	951	951
STM	Pearson Correlation	.060	.969**	.264**	1.000	.208**
	Sig. (2-tailed)	.065	.000	.000	.	.000
	N	950	951	951	951	951
LACTM	Pearson Correlation	.085**	.196**	.091**	.208**	1.000
	Sig. (2-tailed)	.008	.000	.005	.000	.
	N	974	951	951	951	975
YEASTM	Pearson Correlation	-.006	.012	.014	.003	.166**
	Sig. (2-tailed)	.862	.715	.662	.928	.000
	N	937	921	921	921	938
SM	Pearson Correlation	.031	.072*	.006	.063	.015
	Sig. (2-tailed)	.290	.028	.850	.056	.634
	N	1167	926	926	926	949
SS	Pearson Correlation	.001	.099**	.058	.074*	.009
	Sig. (2-tailed)	.982	.003	.077	.024	.777
	N	1167	926	926	926	949
ST	Pearson Correlation	.022	.082*	-.011	.072*	.007
	Sig. (2-tailed)	.448	.013	.746	.028	.834
	N	1167	926	926	926	949
LACT	Pearson Correlation	.049	-.028	.005	-.034	.055
	Sig. (2-tailed)	.094	.395	.869	.304	.088
	N	1185	939	939	939	963
YEAST	Pearson Correlation	.053	-.018	.075*	-.015	.035
	Sig. (2-tailed)	.071	.584	.023	.657	.285
	N	1156	918	918	918	938
SEX	Pearson Correlation	.029	-.026	.004	-.018	.002
	Sig. (2-tailed)	.327	.426	.893	.586	.953
	N	1179	923	923	923	946
HIGHRISK	Pearson Correlation	-.275**	.012	-.023	.010	-.076*
	Sig. (2-tailed)	.000	.733	.493	.777	.024
	N	1170	865	865	865	887
WTCENT	Pearson Correlation	-.069*	-.055	-.118**	-.058	-.036
	Sig. (2-tailed)	.026	.122	.001	.102	.306
	N	1021	804	804	804	824
HTCENT	Pearson Correlation	-.041	.040	-.029	.033	-.064
	Sig. (2-tailed)	.193	.255	.415	.344	.067
	N	1026	812	812	812	830
WTCENTA	Pearson Correlation	-.084**	.047	-.010	.049	.038
	Sig. (2-tailed)	.005	.159	.770	.142	.255
	N	1133	890	890	890	912
HTCENTA	Pearson Correlation	-.041	.042	-.082*	.035	-.005
	Sig. (2-tailed)	.169	.210	.015	.298	.870
	N	1113	877	877	877	899

Correlations

		DEPCAT	SMM	SSM	STM	LACTM
WTCENTB	Pearson Correlation	-.047	.005	-.060	-.010	.023
	Sig. (2-tailed)	.112	.882	.073	.762	.485
	N	1131	891	891	891	914
HTCENTB	Pearson Correlation	-.062*	.023	.012	.020	.016
	Sig. (2-tailed)	.036	.497	.716	.551	.639
	N	1126	886	886	886	909
CIMMUN	Pearson Correlation	.013	-.008	.006	.000	-.017
	Sig. (2-tailed)	.668	.818	.859	.994	.600
	N	1144	898	898	898	919
MEDICAT	Pearson Correlation	-.076**	.030	-.002	.029	-.045
	Sig. (2-tailed)	.010	.372	.940	.386	.175
	N	1151	901	901	901	923
AGEWEAN	Pearson Correlation	-.162**	.011	-.017	-.004	-.022
	Sig. (2-tailed)	.000	.737	.619	.912	.508
	N	1146	901	901	901	923
BRFEED	Pearson Correlation	.287**	.027	.102**	.051	.092**
	Sig. (2-tailed)	.000	.410	.002	.127	.005
	N	1163	913	913	913	934
DUMMY	Pearson Correlation	-.173**	.052	.023	.061	.013
	Sig. (2-tailed)	.000	.139	.512	.083	.706
	N	1026	805	805	805	826
STILLDMY	Pearson Correlation	-.107**	.046	.009	.048	-.085*
	Sig. (2-tailed)	.003	.253	.830	.237	.033
	N	779	611	611	611	624
VITAMIN	Pearson Correlation	.131**	.008	.073*	.017	.082*
	Sig. (2-tailed)	.000	.819	.034	.620	.016
	N	1079	851	851	851	874
SIBLINGS	Pearson Correlation	-.011	.058	-.015	.075*	.086**
	Sig. (2-tailed)	.698	.083	.655	.023	.008
	N	1163	911	911	911	933
MUM_AGE	Pearson Correlation	-.313**	.023	-.016	.013	.000
	Sig. (2-tailed)	.000	.492	.636	.693	.996
	N	1121	881	881	881	903
MARSTAT	Pearson Correlation	.371**	.052	.025	.055	.087*
	Sig. (2-tailed)	.000	.137	.469	.116	.011
	N	1060	831	831	831	852
SC	Pearson Correlation	.427**	.028	.017	.025	.092*
	Sig. (2-tailed)	.000	.451	.640	.509	.012
	N	930	726	726	726	745
SMOKE	Pearson Correlation	-.251**	-.086*	-.068	-.082*	-.170**
	Sig. (2-tailed)	.000	.016	.054	.021	.000
	N	1013	791	791	791	810
EMPLOY	Pearson Correlation	.278**	.005	.027	.017	.126**
	Sig. (2-tailed)	.000	.881	.409	.601	.000
	N	1158	910	910	910	932
BMEAL	Pearson Correlation	.021	.045	.004	.042	.071*
	Sig. (2-tailed)	.474	.175	.913	.207	.030
	N	1158	924	924	924	948
DMEAL	Pearson Correlation	.058*	.033	-.016	.029	.011
	Sig. (2-tailed)	.048	.314	.623	.378	.726
	N	1158	925	925	925	949

Correlations

		DEPCAT	SMM	SSM	STM	LACTM
TMEAL	Pearson Correlation	.020	-.011	-.030	-.014	-.048
	Sig. (2-tailed)	.503	.737	.364	.673	.139
	N	1157	925	925	925	949
SMEAL	Pearson Correlation	-.125**	-.019	.009	-.011	-.070*
	Sig. (2-tailed)	.000	.569	.788	.731	.031
	N	1155	923	923	923	947
NMEAL	Pearson Correlation	.006	.033	.009	.034	-.040
	Sig. (2-tailed)	.842	.314	.788	.302	.218
	N	1156	922	922	922	946
BVESSEL	Pearson Correlation	-.143**	.001	-.059	-.012	-.047
	Sig. (2-tailed)	.000	.974	.077	.708	.146
	N	1146	914	914	914	938
FVESSEL	Pearson Correlation	.113**	.061	.032	.062	.021
	Sig. (2-tailed)	.000	.064	.341	.059	.514
	N	1144	912	912	912	936
CVESSEL	Pearson Correlation	-.078**	-.063	.022	-.055	-.005
	Sig. (2-tailed)	.008	.059	.501	.096	.879
	N	1144	913	913	913	937
SNACK	Pearson Correlation	-.026	.025	.004	.015	-.044
	Sig. (2-tailed)	.390	.463	.894	.666	.183
	N	1116	888	888	888	911
BSNACK	Pearson Correlation	.063*	-.007	.008	-.006	-.066*
	Sig. (2-tailed)	.039	.827	.808	.854	.048
	N	1086	861	861	861	885
SSNACK	Pearson Correlation	-.097**	-.045	.023	-.034	.011
	Sig. (2-tailed)	.001	.188	.503	.322	.745
	N	1085	860	860	860	884
CSNACK	Pearson Correlation	-.187**	.044	-.018	.049	-.001
	Sig. (2-tailed)	.000	.202	.596	.150	.969
	N	1086	861	861	861	885
FSNACK	Pearson Correlation	-.033	.001	-.035	.004	-.002
	Sig. (2-tailed)	.279	.967	.303	.915	.945
	N	1084	860	860	860	884
TOOTHPAS	Pearson Correlation	.058	.029	.020	.028	.028
	Sig. (2-tailed)	.061	.393	.567	.418	.412
	N	1065	848	848	848	872
FLUOR	Pearson Correlation	-.003	-.068*	-.042	-.067*	-.036
	Sig. (2-tailed)	.910	.039	.211	.044	.275
	N	1137	905	905	905	929
CARE	Pearson Correlation	-.006	.063	.024	.059	.041
	Sig. (2-tailed)	.848	.059	.465	.076	.205
	N	1140	910	910	910	933
SHOPTYPE	Pearson Correlation	-.166**	-.054	.010	-.051	-.047
	Sig. (2-tailed)	.000	.099	.755	.124	.147
	N	1155	921	921	921	945
BEDTIME	Pearson Correlation	-.155**	-.023	.010	-.023	-.081*
	Sig. (2-tailed)	.000	.490	.756	.484	.013
	N	1147	915	915	915	939

Correlations

		DEPCAT	SMM	SSM	STM	LACTM
NIGHT	Pearson Correlation	-.102**	-.023	-.017	-.028	-.039
	Sig. (2-tailed)	.001	.486	.616	.396	.231
	N	1126	902	902	902	925
WHOBRUSH	Pearson Correlation	.111**	.020	.053	.025	.030
	Sig. (2-tailed)	.000	.545	.114	.458	.361
	N	1120	896	896	896	919

Correlations

		YEASTM	SM	SS	ST	LACT
DEPCAT	Pearson Correlation	-.006	.031	.001	.022	.049
	Sig. (2-tailed)	.862	.290	.982	.448	.094
	N	937	1167	1167	1167	1185
SMM	Pearson Correlation	.012	.072*	.099**	.082*	-.028
	Sig. (2-tailed)	.715	.028	.003	.013	.395
	N	921	926	926	926	939
SSM	Pearson Correlation	.014	.006	.058	-.011	.005
	Sig. (2-tailed)	.662	.850	.077	.746	.869
	N	921	926	926	926	939
STM	Pearson Correlation	.003	.063	.074*	.072*	-.034
	Sig. (2-tailed)	.928	.056	.024	.028	.304
	N	921	926	926	926	939
LACTM	Pearson Correlation	.166**	.015	.009	.007	.055
	Sig. (2-tailed)	.000	.634	.777	.834	.088
	N	938	949	949	949	963
YEASTM	Pearson Correlation	1.000	-.011	-.033	-.026	.015
	Sig. (2-tailed)	.	.733	.323	.425	.659
	N	938	914	914	914	927
SM	Pearson Correlation	-.011	1.000	.132**	.964**	.025
	Sig. (2-tailed)	.733	.	.000	.000	.401
	N	914	1168	1168	1168	1168
SS	Pearson Correlation	-.033	.132**	1.000	.266**	-.022
	Sig. (2-tailed)	.323	.000	.	.000	.462
	N	914	1168	1168	1168	1168
ST	Pearson Correlation	-.026	.964**	.266**	1.000	.022
	Sig. (2-tailed)	.425	.000	.000	.	.455
	N	914	1168	1168	1168	1168
LACT	Pearson Correlation	.015	.025	-.022	.022	1.000
	Sig. (2-tailed)	.659	.401	.462	.455	.
	N	927	1168	1168	1168	1186
YEAST	Pearson Correlation	.098**	.026	-.035	.021	.067*
	Sig. (2-tailed)	.003	.386	.236	.469	.022
	N	905	1144	1144	1144	1157
SEX	Pearson Correlation	.000	-.022	.032	-.027	.033
	Sig. (2-tailed)	.998	.456	.284	.362	.267
	N	912	1135	1135	1135	1153
HIGHRISK	Pearson Correlation	.000	-.098**	-.051	-.098**	-.028
	Sig. (2-tailed)	.994	.001	.094	.001	.362
	N	854	1061	1061	1061	1079
WTCENT	Pearson Correlation	.011	-.016	.052	-.001	.004
	Sig. (2-tailed)	.767	.619	.105	.981	.912
	N	799	982	982	982	997
HTCENT	Pearson Correlation	-.068	.065*	.028	.078*	.022
	Sig. (2-tailed)	.055	.040	.380	.014	.478
	N	804	989	989	989	1004
WTCENTA	Pearson Correlation	-.035	.045	.047	.050	-.039
	Sig. (2-tailed)	.300	.135	.118	.097	.194
	N	881	1090	1090	1090	1108
HTCENTA	Pearson Correlation	-.053	.034	.044	.038	-.019
	Sig. (2-tailed)	.121	.272	.146	.209	.534
	N	869	1070	1070	1070	1088

Correlations

		YEASTM	SM	SS	ST	LACT
WTCENTB	Pearson Correlation	-.012	.025	.042	.038	-.062*
	Sig. (2-tailed)	.730	.416	.167	.212	.039
	N	881	1091	1091	1091	1109
HTCENTB	Pearson Correlation	-.029	.022	.038	.025	-.009
	Sig. (2-tailed)	.390	.471	.213	.407	.777
	N	876	1086	1086	1086	1104
CIMMUN	Pearson Correlation	.045	.014	-.018	.010	.002
	Sig. (2-tailed)	.177	.650	.544	.731	.956
	N	885	1101	1101	1101	1119
MEDICAT	Pearson Correlation	-.071*	-.011	-.014	-.020	.018
	Sig. (2-tailed)	.033	.715	.641	.510	.542
	N	890	1109	1109	1109	1127
AGEWEAN	Pearson Correlation	.003	-.019	-.026	-.028	-.001
	Sig. (2-tailed)	.940	.534	.379	.350	.986
	N	889	1106	1106	1106	1124
BRFEED	Pearson Correlation	-.002	.000	-.011	.003	.037
	Sig. (2-tailed)	.963	.993	.718	.910	.209
	N	900	1121	1121	1121	1139
DUMMY	Pearson Correlation	-.005	.077*	.055	.082*	.011
	Sig. (2-tailed)	.896	.015	.085	.010	.725
	N	795	990	990	990	1007
STILLDMY	Pearson Correlation	-.017	.006	.014	.016	-.004
	Sig. (2-tailed)	.682	.861	.698	.660	.912
	N	600	751	751	751	765
VITAMIN	Pearson Correlation	.048	.017	.004	.011	.004
	Sig. (2-tailed)	.165	.584	.892	.723	.892
	N	843	1044	1044	1044	1060
SIBLINGS	Pearson Correlation	.059	.066*	.012	.071*	.047
	Sig. (2-tailed)	.079	.028	.680	.018	.110
	N	900	1121	1121	1121	1138
MUM_AGE	Pearson Correlation	-.009	.054	.035	.073*	-.008
	Sig. (2-tailed)	.796	.078	.248	.016	.788
	N	870	1078	1078	1078	1096
MARSTAT	Pearson Correlation	-.045	.039	-.015	.031	.024
	Sig. (2-tailed)	.194	.216	.639	.325	.447
	N	820	1018	1018	1018	1035
SC	Pearson Correlation	.005	.013	-.001	.010	.042
	Sig. (2-tailed)	.889	.702	.971	.764	.200
	N	716	897	897	897	912
SMOKE	Pearson Correlation	-.047	-.080*	.031	-.082**	-.047
	Sig. (2-tailed)	.185	.012	.324	.010	.142
	N	783	981	981	981	992
EMPLOY	Pearson Correlation	.031	.049	-.029	.048	.054
	Sig. (2-tailed)	.347	.104	.326	.112	.067
	N	898	1116	1116	1116	1134
BMEAL	Pearson Correlation	-.011	-.045	-.014	-.046	.007
	Sig. (2-tailed)	.730	.134	.648	.127	.817
	N	913	1124	1124	1124	1141
DMEAL	Pearson Correlation	.045	.061*	-.016	.058	-.005
	Sig. (2-tailed)	.175	.042	.585	.052	.865
	N	914	1124	1124	1124	1141

Correlations

		YEASTM	SM	SS	ST	LACT
TMEAL	Pearson Correlation	-.038	.040	-.012	.039	.017
	Sig. (2-tailed)	.248	.175	.696	.196	.568
	N	914	1123	1123	1123	1140
SMEAL	Pearson Correlation	-.010	-.040	.014	-.028	-.034
	Sig. (2-tailed)	.768	.179	.639	.342	.249
	N	912	1122	1122	1122	1139
NMEAL	Pearson Correlation	-.095**	.010	.003	.010	.006
	Sig. (2-tailed)	.004	.736	.917	.731	.834
	N	911	1122	1122	1122	1139
BVESSEL	Pearson Correlation	-.005	-.103**	.009	-.091**	.002
	Sig. (2-tailed)	.877	.001	.774	.003	.955
	N	903	1112	1112	1112	1129
FVESSEL	Pearson Correlation	.056	.051	-.002	.044	.032
	Sig. (2-tailed)	.093	.088	.940	.147	.290
	N	901	1110	1110	1110	1127
CVESSEL	Pearson Correlation	-.009	.024	.002	.027	.016
	Sig. (2-tailed)	.795	.424	.952	.364	.595
	N	902	1111	1111	1111	1128
SNACK	Pearson Correlation	-.009	-.002	-.011	.003	-.032
	Sig. (2-tailed)	.780	.957	.717	.912	.289
	N	876	1082	1082	1082	1099
BSNACK	Pearson Correlation	-.024	.007	-.037	-.009	-.008
	Sig. (2-tailed)	.477	.818	.231	.762	.803
	N	851	1053	1053	1053	1069
SSNACK	Pearson Correlation	-.022	.001	-.024	.006	-.038
	Sig. (2-tailed)	.521	.985	.441	.846	.213
	N	850	1052	1052	1052	1068
CSNACK	Pearson Correlation	-.013	-.040	-.011	-.030	-.045
	Sig. (2-tailed)	.698	.193	.712	.325	.140
	N	851	1053	1053	1053	1069
FSNACK	Pearson Correlation	-.015	.060	.000	.073*	-.006
	Sig. (2-tailed)	.658	.053	.995	.018	.845
	N	850	1051	1051	1051	1067
TOOTHPAS	Pearson Correlation	-.021	.004	.027	.019	.083**
	Sig. (2-tailed)	.543	.897	.386	.532	.007
	N	839	1032	1032	1032	1048
FLUOR	Pearson Correlation	.014	-.078**	-.028	-.080**	-.004
	Sig. (2-tailed)	.683	.009	.351	.007	.901
	N	894	1105	1105	1105	1121
CARE	Pearson Correlation	.033	.037	.024	.039	.048
	Sig. (2-tailed)	.318	.218	.422	.193	.105
	N	899	1106	1106	1106	1123
SHOPTYPE	Pearson Correlation	.056	-.037	.015	-.044	.019
	Sig. (2-tailed)	.093	.210	.613	.144	.517
	N	910	1121	1121	1121	1138
BEDTIME	Pearson Correlation	-.057	-.061*	.056	-.055	-.009
	Sig. (2-tailed)	.086	.042	.062	.067	.754
	N	904	1113	1113	1113	1130

Correlations

		YEASTM	SM	SS	ST	LACT
NIGHT	Pearson Correlation	.010	-.048	.032	-.037	-.077*
	Sig. (2-tailed)	.776	.109	.293	.218	.010
	N	890	1094	1094	1094	1110
WHOBRUSH	Pearson Correlation	-.014	.002	-.004	.009	.097**
	Sig. (2-tailed)	.668	.935	.906	.770	.001
	N	886	1088	1088	1088	1105

Correlations

		YEAST	SEX	HIGHRISK	WTCENT	HTCENT
DEPCAT	Pearson Correlation	.053	.029	-.275**	-.069*	-.041
	Sig. (2-tailed)	.071	.327	.000	.026	.193
	N	1156	1179	1170	1021	1026
SMM	Pearson Correlation	-.018	-.026	.012	-.055	.040
	Sig. (2-tailed)	.584	.426	.733	.122	.255
	N	918	923	865	804	812
SSM	Pearson Correlation	.075*	.004	-.023	-.118**	-.029
	Sig. (2-tailed)	.023	.893	.493	.001	.415
	N	918	923	865	804	812
STM	Pearson Correlation	-.015	-.018	.010	-.058	.033
	Sig. (2-tailed)	.657	.586	.777	.102	.344
	N	918	923	865	804	812
LACTM	Pearson Correlation	.035	.002	-.076*	-.036	-.064
	Sig. (2-tailed)	.285	.953	.024	.306	.067
	N	938	946	887	824	830
YEASTM	Pearson Correlation	.098**	.000	.000	.011	-.068
	Sig. (2-tailed)	.003	.998	.994	.767	.055
	N	905	912	854	799	804
SM	Pearson Correlation	.026	-.022	-.098**	-.016	.065*
	Sig. (2-tailed)	.386	.456	.001	.619	.040
	N	1144	1135	1061	982	989
SS	Pearson Correlation	-.035	.032	-.051	.052	.028
	Sig. (2-tailed)	.236	.284	.094	.105	.380
	N	1144	1135	1061	982	989
ST	Pearson Correlation	.021	-.027	-.098**	-.001	.078*
	Sig. (2-tailed)	.469	.362	.001	.981	.014
	N	1144	1135	1061	982	989
LACT	Pearson Correlation	.067*	.033	-.028	.004	.022
	Sig. (2-tailed)	.022	.267	.362	.912	.478
	N	1157	1153	1079	997	1004
YEAST	Pearson Correlation	1.000	-.004	-.030	.048	.038
	Sig. (2-tailed)	.	.901	.323	.133	.230
	N	1157	1125	1053	974	983
SEX	Pearson Correlation	-.004	1.000	.016	-.068*	-.223**
	Sig. (2-tailed)	.901	.	.601	.030	.000
	N	1125	1180	1087	1021	1027
HIGHRISK	Pearson Correlation	-.030	.016	1.000	-.008	-.005
	Sig. (2-tailed)	.323	.601	.	.801	.888
	N	1053	1087	1171	940	945
WTCENT	Pearson Correlation	.048	-.068*	-.008	1.000	.382**
	Sig. (2-tailed)	.133	.030	.801	.	.000
	N	974	1021	940	1021	936
HTCENT	Pearson Correlation	.038	-.223**	-.005	.382**	1.000
	Sig. (2-tailed)	.230	.000	.888	.000	.
	N	983	1027	945	936	1027
WTCENTA	Pearson Correlation	.030	.001	.072*	.330**	.247**
	Sig. (2-tailed)	.329	.970	.020	.000	.000
	N	1083	1134	1044	988	993
HTCENTA	Pearson Correlation	.016	-.001	.028	.287**	.332**
	Sig. (2-tailed)	.600	.964	.365	.000	.000
	N	1064	1114	1025	970	978

Correlations

		YEAST	SEX	HIGHRISK	WTCENT	HTCENT
WTCENTB	Pearson Correlation	.039	-.057	.071*	.158**	.152**
	Sig. (2-tailed)	.196	.055	.022	.000	.000
	N	1082	1132	1047	983	988
HTCENTB	Pearson Correlation	.037	.052	.045	.175**	.191**
	Sig. (2-tailed)	.228	.082	.148	.000	.000
	N	1077	1127	1042	979	984
CIMMUN	Pearson Correlation	-.035	-.012	-.098**	.058	.003
	Sig. (2-tailed)	.243	.692	.002	.068	.932
	N	1094	1145	1053	990	998
MEDICAT	Pearson Correlation	-.072*	.030	.000	.035	-.019
	Sig. (2-tailed)	.016	.315	.997	.272	.546
	N	1100	1152	1062	994	1001
AGEWEAN	Pearson Correlation	-.023	.010	.211**	-.026	-.047
	Sig. (2-tailed)	.453	.727	.000	.413	.139
	N	1097	1147	1058	993	998
BRFEED	Pearson Correlation	.016	.015	-.275**	-.079*	.017
	Sig. (2-tailed)	.586	.610	.000	.012	.584
	N	1112	1164	1074	1006	1015
DUMMY	Pearson Correlation	-.067*	.022	.147**	-.023	.025
	Sig. (2-tailed)	.037	.491	.000	.489	.457
	N	980	1027	950	886	892
STILLDMY	Pearson Correlation	-.036	-.014	.136**	-.059	-.015
	Sig. (2-tailed)	.331	.696	.000	.130	.690
	N	744	780	727	671	670
VITAMIN	Pearson Correlation	.024	.025	-.171**	-.004	-.004
	Sig. (2-tailed)	.437	.408	.000	.903	.891
	N	1034	1079	1001	930	933
SIBLINGS	Pearson Correlation	-.029	-.017	-.078*	.069*	.048
	Sig. (2-tailed)	.327	.573	.011	.028	.124
	N	1111	1164	1072	1008	1014
MUM_AGE	Pearson Correlation	-.048	-.031	.204**	.051	-.002
	Sig. (2-tailed)	.117	.292	.000	.109	.940
	N	1069	1122	1034	978	981
MARSTAT	Pearson Correlation	.058	.010	-.376**	-.021	-.021
	Sig. (2-tailed)	.063	.754	.000	.516	.529
	N	1010	1060	983	917	930
SC	Pearson Correlation	-.011	-.023	-.299**	-.009	.002
	Sig. (2-tailed)	.741	.486	.000	.804	.950
	N	888	931	862	813	816
SMOKE	Pearson Correlation	-.049	.025	.308**	.064	.045
	Sig. (2-tailed)	.131	.428	.000	.059	.181
	N	968	1014	948	874	878
EMPLOY	Pearson Correlation	.069*	-.006	-.316**	-.001	-.013
	Sig. (2-tailed)	.022	.836	.000	.975	.687
	N	1109	1159	1069	1004	1009
BMEAL	Pearson Correlation	.003	.028	-.089**	-.047	-.014
	Sig. (2-tailed)	.913	.348	.004	.140	.670
	N	1113	1133	1057	983	985
DMEAL	Pearson Correlation	-.034	.008	-.143**	-.055	.016
	Sig. (2-tailed)	.252	.781	.000	.087	.618
	N	1113	1133	1057	983	986

Correlations

		YEAST	SEX	HIGHRISK	WTCENT	HTCENT
TMEAL	Pearson Correlation	-.012	.047	.007	-.045	.056
	Sig. (2-tailed)	.693	.112	.812	.158	.078
	N	1112	1132	1056	982	985
SMEAL	Pearson Correlation	.021	-.002	.047	.051	.055
	Sig. (2-tailed)	.493	.955	.126	.108	.087
	N	1111	1132	1056	982	985
NMEAL	Pearson Correlation	-.088**	-.031	.052	.[a]	.057
	Sig. (2-tailed)	.003	.291	.091	.	.072
	N	1111	1134	1057	983	986
BVESSEL	Pearson Correlation	-.051	.044	.198**	.060	.077*
	Sig. (2-tailed)	.092	.140	.000	.063	.016
	N	1101	1121	1046	972	974
FVESSEL	Pearson Correlation	.069*	-.019	-.189**	-.054	.004
	Sig. (2-tailed)	.021	.528	.000	.092	.903
	N	1099	1120	1045	971	973
CVESSEL	Pearson Correlation	.001	-.034	.061*	.021	-.032
	Sig. (2-tailed)	.985	.252	.049	.506	.317
	N	1100	1119	1044	970	972
SNACK	Pearson Correlation	.005	-.040	.077*	-.012	.061
	Sig. (2-tailed)	.863	.188	.013	.722	.060
	N	1071	1091	1020	948	951
BSNACK	Pearson Correlation	.038	.022	.047	-.040	.036
	Sig. (2-tailed)	.221	.478	.140	.220	.277
	N	1042	1062	994	921	924
SSNACK	Pearson Correlation	-.068*	.004	.141**	-.036	-.008
	Sig. (2-tailed)	.029	.902	.000	.274	.819
	N	1041	1061	993	920	923
CSNACK	Pearson Correlation	-.007	-.001	.186**	-.012	.010
	Sig. (2-tailed)	.819	.982	.000	.706	.764
	N	1042	1062	994	921	924
FSNACK	Pearson Correlation	.018	-.068*	-.082**	.002	-.027
	Sig. (2-tailed)	.568	.027	.009	.953	.417
	N	1040	1060	993	920	923
TOOTHPAS	Pearson Correlation	-.008	.005	-.158**	-.035	-.050
	Sig. (2-tailed)	.791	.872	.000	.288	.130
	N	1021	1045	973	912	909
FLUOR	Pearson Correlation	-.045	-.007	.084**	.016	-.001
	Sig. (2-tailed)	.138	.826	.006	.614	.974
	N	1094	1115	1042	964	968
CARE	Pearson Correlation	.025	-.043	-.045	.006	-.010
	Sig. (2-tailed)	.400	.154	.151	.859	.761
	N	1095	1117	1042	968	971
SHOPTYPE	Pearson Correlation	-.014	.006	.123**	.004	-.027
	Sig. (2-tailed)	.647	.842	.000	.905	.394
	N	1110	1130	1055	978	983
BEDTIME	Pearson Correlation	.013	.003	.116**	.059	.067*
	Sig. (2-tailed)	.671	.917	.000	.064	.036
	N	1103	1123	1048	971	976

Correlations

		YEAST	SEX	HIGHRISK	WTCENT	HTCENT
NIGHT	Pearson Correlation	-.028	.019	.065*	.014	-.010
	Sig. (2-tailed)	.360	.537	.036	.664	.762
	N	1083	1102	1030	951	960
WHOBRUSH	Pearson Correlation	-.004	.029	-.199**	-.039	-.086**
	Sig. (2-tailed)	.899	.334	.000	.225	.008
	N	1077	1095	1022	947	955

Correlations

		WTCENTA	HTCENTA	WTCENTB	HTCENTB	CIMMUN
DEPCAT	Pearson Correlation	-.084**	-.041	-.047	-.062*	.013
	Sig. (2-tailed)	.005	.169	.112	.036	.668
	N	1133	1113	1131	1126	1144
SMM	Pearson Correlation	.047	.042	.005	.023	-.008
	Sig. (2-tailed)	.159	.210	.882	.497	.818
	N	890	877	891	886	898
SSM	Pearson Correlation	-.010	-.082*	-.060	.012	.006
	Sig. (2-tailed)	.770	.015	.073	.716	.859
	N	890	877	891	886	898
STM	Pearson Correlation	.049	.035	-.010	.020	.000
	Sig. (2-tailed)	.142	.298	.762	.551	.994
	N	890	877	891	886	898
LACTM	Pearson Correlation	.038	-.005	.023	.016	-.017
	Sig. (2-tailed)	.255	.870	.485	.639	.600
	N	912	899	914	909	919
YEASTM	Pearson Correlation	-.035	-.053	-.012	-.029	.045
	Sig. (2-tailed)	.300	.121	.730	.390	.177
	N	881	869	881	876	885
SM	Pearson Correlation	.045	.034	.025	.022	.014
	Sig. (2-tailed)	.135	.272	.416	.471	.650
	N	1090	1070	1091	1086	1101
SS	Pearson Correlation	.047	.044	.042	.038	-.018
	Sig. (2-tailed)	.118	.146	.167	.213	.544
	N	1090	1070	1091	1086	1101
ST	Pearson Correlation	.050	.038	.038	.025	.010
	Sig. (2-tailed)	.097	.209	.212	.407	.731
	N	1090	1070	1091	1086	1101
LACT	Pearson Correlation	-.039	-.019	-.062*	-.009	.002
	Sig. (2-tailed)	.194	.534	.039	.777	.956
	N	1108	1088	1109	1104	1119
YEAST	Pearson Correlation	.030	.016	.039	.037	-.035
	Sig. (2-tailed)	.329	.600	.196	.228	.243
	N	1083	1064	1082	1077	1094
SEX	Pearson Correlation	.001	-.001	-.057	.052	-.012
	Sig. (2-tailed)	.970	.964	.055	.082	.692
	N	1134	1114	1132	1127	1145
HIGHRISK	Pearson Correlation	.072*	.028	.071*	.045	-.098**
	Sig. (2-tailed)	.020	.365	.022	.148	.002
	N	1044	1025	1047	1042	1053
WTCENT	Pearson Correlation	.330**	.287**	.158**	.175**	.058
	Sig. (2-tailed)	.000	.000	.000	.000	.068
	N	988	970	983	979	990
HTCENT	Pearson Correlation	.247**	.332**	.152**	.191**	.003
	Sig. (2-tailed)	.000	.000	.000	.000	.932
	N	993	978	988	984	998
WTCENTA	Pearson Correlation	1.000	.522**	.412**	.351**	.044
	Sig. (2-tailed)	.	.000	.000	.000	.141
	N	1134	1111	1101	1095	1105
HTCENTA	Pearson Correlation	.522**	1.000	.321**	.460**	.016
	Sig. (2-tailed)	.000	.	.000	.000	.589
	N	1111	1114	1082	1079	1086

Correlations

		WTCENTA	HTCENTA	WTCENTB	HTCENTB	CIMMUN
WTCENTB	Pearson Correlation	.412**	.321**	1.000	.407**	-.020
	Sig. (2-tailed)	.000	.000	.	.000	.510
	N	1101	1082	1132	1123	1102
HTCENTB	Pearson Correlation	.351**	.460**	.407**	1.000	-.014
	Sig. (2-tailed)	.000	.000	.000	.	.634
	N	1095	1079	1123	1127	1097
CIMMUN	Pearson Correlation	.044	.016	-.020	-.014	1.000
	Sig. (2-tailed)	.141	.589	.510	.634	.
	N	1105	1086	1102	1097	1145
MEDICAT	Pearson Correlation	.133**	.052	.070*	.058	-.040
	Sig. (2-tailed)	.000	.085	.019	.053	.179
	N	1107	1087	1106	1101	1122
AGEWEAN	Pearson Correlation	-.072*	-.073*	-.026	-.012	-.031
	Sig. (2-tailed)	.017	.016	.397	.682	.296
	N	1102	1082	1101	1096	1116
BRFEED	Pearson Correlation	-.064*	-.101**	-.054	-.062*	.054
	Sig. (2-tailed)	.031	.001	.070	.038	.071
	N	1118	1098	1117	1112	1132
DUMMY	Pearson Correlation	.042	.034	-.058	.018	.050
	Sig. (2-tailed)	.184	.293	.070	.574	.113
	N	988	969	989	983	1000
STILLDMY	Pearson Correlation	.041	.000	.006	.070	.029
	Sig. (2-tailed)	.264	.997	.879	.054	.429
	N	751	741	749	747	760
VITAMIN	Pearson Correlation	.076*	.028	.020	.049	.048
	Sig. (2-tailed)	.015	.368	.521	.119	.119
	N	1036	1017	1036	1031	1048
SIBLINGS	Pearson Correlation	.056	.008	-.033	-.052	.092**
	Sig. (2-tailed)	.062	.799	.277	.086	.002
	N	1120	1100	1116	1111	1133
MUM_AGE	Pearson Correlation	.002	.005	-.014	.016	.023
	Sig. (2-tailed)	.958	.882	.637	.608	.454
	N	1077	1058	1077	1072	1092
MARSTAT	Pearson Correlation	-.038	-.015	-.045	-.020	.043
	Sig. (2-tailed)	.222	.646	.151	.526	.163
	N	1019	1002	1015	1011	1032
SC	Pearson Correlation	-.042	.004	-.040	-.097**	.004
	Sig. (2-tailed)	.212	.896	.226	.004	.901
	N	897	887	902	900	903
SMOKE	Pearson Correlation	.013	.060	.039	.046	-.076*
	Sig. (2-tailed)	.685	.063	.225	.152	.017
	N	975	954	977	972	986
EMPLOY	Pearson Correlation	-.036	-.052	-.066*	-.064*	.041
	Sig. (2-tailed)	.228	.087	.029	.033	.169
	N	1115	1095	1113	1108	1126
BMEAL	Pearson Correlation	-.001	-.051	-.045	-.032	.011
	Sig. (2-tailed)	.981	.092	.137	.299	.722
	N	1090	1072	1089	1085	1099
DMEAL	Pearson Correlation	-.023	.000	-.013	.000	.001
	Sig. (2-tailed)	.443	.995	.672	.995	.975
	N	1090	1072	1089	1085	1099

Correlations

		WTCENTA	HTCENTA	WTCENTB	HTCENTB	CIMMUN
TMEAL	Pearson Correlation	-.016	.002	-.007	.041	-.021
	Sig. (2-tailed)	.591	.954	.817	.177	.493
	N	1089	1071	1088	1084	1098
SMEAL	Pearson Correlation	.004	.048	.018	.011	-.027
	Sig. (2-tailed)	.883	.116	.552	.717	.367
	N	1089	1071	1088	1084	1098
NMEAL	Pearson Correlation	.066*	.070*	-.012	-.012	.006
	Sig. (2-tailed)	.030	.022	.682	.698	.844
	N	1091	1073	1090	1086	1100
BVESSEL	Pearson Correlation	.062*	.039	.025	.046	-.020
	Sig. (2-tailed)	.041	.203	.415	.136	.510
	N	1078	1060	1078	1074	1087
FVESSEL	Pearson Correlation	-.035	.014	-.022	-.030	.057
	Sig. (2-tailed)	.256	.647	.464	.329	.060
	N	1077	1059	1077	1073	1086
CVESSEL	Pearson Correlation	-.067*	-.060	-.009	-.026	-.060*
	Sig. (2-tailed)	.028	.050	.763	.389	.050
	N	1076	1058	1076	1072	1085
SNACK	Pearson Correlation	-.028	-.022	.035	-.028	-.024
	Sig. (2-tailed)	.365	.478	.263	.361	.440
	N	1049	1031	1049	1046	1058
BSNACK	Pearson Correlation	-.003	.017	.012	-.003	-.018
	Sig. (2-tailed)	.930	.590	.703	.916	.557
	N	1022	1005	1022	1018	1031
SSNACK	Pearson Correlation	-.008	-.039	-.009	-.037	-.060
	Sig. (2-tailed)	.802	.219	.769	.241	.056
	N	1021	1004	1021	1017	1030
CSNACK	Pearson Correlation	.032	.022	.004	-.009	-.024
	Sig. (2-tailed)	.300	.480	.909	.777	.435
	N	1022	1005	1022	1018	1031
FSNACK	Pearson Correlation	-.002	-.025	-.063*	-.090**	.023
	Sig. (2-tailed)	.957	.432	.043	.004	.463
	N	1021	1004	1021	1017	1029
TOOTHPAS	Pearson Correlation	-.102**	-.114**	-.091**	-.105**	.018
	Sig. (2-tailed)	.001	.000	.004	.001	.569
	N	1007	992	1004	1001	1012
FLUOR	Pearson Correlation	.028	-.008	-.002	-.038	.008
	Sig. (2-tailed)	.355	.798	.955	.220	.783
	N	1072	1056	1071	1067	1081
CARE	Pearson Correlation	.030	.030	.027	-.026	-.021
	Sig. (2-tailed)	.331	.325	.370	.399	.485
	N	1075	1059	1074	1070	1083
SHOPTYPE	Pearson Correlation	.007	-.056	.041	.005	-.045
	Sig. (2-tailed)	.827	.067	.178	.859	.138
	N	1087	1069	1087	1082	1097
BEDTIME	Pearson Correlation	.075*	.078*	.075*	.024	-.003
	Sig. (2-tailed)	.014	.011	.013	.431	.915
	N	1080	1062	1079	1076	1089

Correlations

		WTCENTA	HTCENTA	WTCENTB	HTCENTB	CIMMUN
NIGHT	Pearson Correlation	.056	-.017	.070*	.024	.009
	Sig. (2-tailed)	.067	.590	.022	.433	.772
	N	1060	1042	1060	1056	1068
WHOBRUSH	Pearson Correlation	-.122**	-.136**	-.132**	-.140**	.010
	Sig. (2-tailed)	.000	.000	.000	.000	.750
	N	1054	1036	1054	1050	1061

Correlations

		MEDICAT	AGEWEAN	BRFEED	DUMMY	STILLDMY
DEPCAT	Pearson Correlation	-.076**	-.162**	.287**	-.173**	-.107**
	Sig. (2-tailed)	.010	.000	.000	.000	.003
	N	1151	1146	1163	1026	779
SMM	Pearson Correlation	.030	.011	.027	.052	.046
	Sig. (2-tailed)	.372	.737	.410	.139	.253
	N	901	901	913	805	611
SSM	Pearson Correlation	-.002	-.017	.102**	.023	.009
	Sig. (2-tailed)	.940	.619	.002	.512	.830
	N	901	901	913	805	611
STM	Pearson Correlation	.029	-.004	.051	.061	.048
	Sig. (2-tailed)	.386	.912	.127	.083	.237
	N	901	901	913	805	611
LACTM	Pearson Correlation	-.045	-.022	.092**	.013	-.085*
	Sig. (2-tailed)	.175	.508	.005	.706	.033
	N	923	923	934	826	624
YEASTM	Pearson Correlation	-.071*	.003	-.002	-.005	-.017
	Sig. (2-tailed)	.033	.940	.963	.896	.682
	N	890	889	900	795	600
SM	Pearson Correlation	-.011	-.019	.000	.077*	.006
	Sig. (2-tailed)	.715	.534	.993	.015	.861
	N	1109	1106	1121	990	751
SS	Pearson Correlation	-.014	-.026	-.011	.055	.014
	Sig. (2-tailed)	.641	.379	.718	.085	.698
	N	1109	1106	1121	990	751
ST	Pearson Correlation	-.020	-.028	.003	.082*	.016
	Sig. (2-tailed)	.510	.350	.910	.010	.660
	N	1109	1106	1121	990	751
LACT	Pearson Correlation	.018	-.001	.037	.011	-.004
	Sig. (2-tailed)	.542	.986	.209	.725	.912
	N	1127	1124	1139	1007	765
YEAST	Pearson Correlation	-.072*	-.023	.016	-.067*	-.036
	Sig. (2-tailed)	.016	.453	.586	.037	.331
	N	1100	1097	1112	980	744
SEX	Pearson Correlation	.030	.010	.015	.022	-.014
	Sig. (2-tailed)	.315	.727	.610	.491	.696
	N	1152	1147	1164	1027	780
HIGHRISK	Pearson Correlation	.000	.211**	-.275**	.147**	.136**
	Sig. (2-tailed)	.997	.000	.000	.000	.000
	N	1062	1058	1074	950	727
WTCENT	Pearson Correlation	.035	-.026	-.079*	-.023	-.059
	Sig. (2-tailed)	.272	.413	.012	.489	.130
	N	994	993	1006	886	671
HTCENT	Pearson Correlation	-.019	-.047	.017	.025	-.015
	Sig. (2-tailed)	.546	.139	.584	.457	.690
	N	1001	998	1015	892	670
WTCENTA	Pearson Correlation	.133**	-.072*	-.064*	.042	.041
	Sig. (2-tailed)	.000	.017	.031	.184	.264
	N	1107	1102	1118	988	751
HTCENTA	Pearson Correlation	.052	-.073*	-.101**	.034	.000
	Sig. (2-tailed)	.085	.016	.001	.293	.997
	N	1087	1082	1098	969	741

Correlations

		MEDICAT	AGEWEAN	BRFEED	DUMMY	STILLDMY
WTCENTB	Pearson Correlation	.070*	-.026	-.054	-.058	.006
	Sig. (2-tailed)	.019	.397	.070	.070	.879
	N	1106	1101	1117	989	749
HTCENTB	Pearson Correlation	.058	-.012	-.062*	.018	.070
	Sig. (2-tailed)	.053	.682	.038	.574	.054
	N	1101	1096	1112	983	747
CIMMUN	Pearson Correlation	-.040	-.031	.054	.050	.029
	Sig. (2-tailed)	.179	.296	.071	.113	.429
	N	1122	1116	1132	1000	760
MEDICAT	Pearson Correlation	1.000	.018	-.002	.027	.060
	Sig. (2-tailed)	.	.536	.940	.394	.095
	N	1152	1122	1139	1004	766
AGEWEAN	Pearson Correlation	.018	1.000	-.241**	.113**	.084*
	Sig. (2-tailed)	.536	.	.000	.000	.021
	N	1122	1147	1139	1010	764
BRFEED	Pearson Correlation	-.002	-.241**	1.000	-.245**	-.172**
	Sig. (2-tailed)	.940	.000	.	.000	.000
	N	1139	1139	1164	1017	771
DUMMY	Pearson Correlation	.027	.113**	-.245**	1.000	.594**
	Sig. (2-tailed)	.394	.000	.000	.	.000
	N	1004	1010	1017	1027	729
STILLDMY	Pearson Correlation	.060	.084*	-.172**	.594**	1.000
	Sig. (2-tailed)	.095	.021	.000	.000	.
	N	766	764	771	729	780
VITAMIN	Pearson Correlation	.038	-.139**	.182**	-.048	-.098**
	Sig. (2-tailed)	.222	.000	.000	.134	.007
	N	1058	1061	1070	970	739
SIBLINGS	Pearson Correlation	-.053	.025	-.003	.095**	.052
	Sig. (2-tailed)	.076	.406	.926	.003	.150
	N	1140	1135	1151	1015	770
MUM_AGE	Pearson Correlation	.046	.175**	-.230**	.146**	.067
	Sig. (2-tailed)	.124	.000	.000	.000	.069
	N	1098	1096	1111	983	748
MARSTAT	Pearson Correlation	-.033	-.168**	.307**	-.125**	-.114**
	Sig. (2-tailed)	.292	.000	.000	.000	.002
	N	1037	1034	1048	928	703
SC	Pearson Correlation	.016	-.159**	.327**	-.201**	-.149**
	Sig. (2-tailed)	.634	.000	.000	.000	.000
	N	912	913	920	816	611
SMOKE	Pearson Correlation	-.015	.213**	-.244**	.152**	.203**
	Sig. (2-tailed)	.647	.000	.000	.000	.000
	N	992	991	1004	888	674
EMPLOY	Pearson Correlation	-.033	-.097**	.218**	-.105**	-.078*
	Sig. (2-tailed)	.261	.001	.000	.001	.031
	N	1133	1131	1146	1014	765
BMEAL	Pearson Correlation	-.025	.046	.038	.001	.017
	Sig. (2-tailed)	.411	.125	.198	.982	.635
	N	1105	1105	1118	989	748
DMEAL	Pearson Correlation	-.041	-.022	.019	.021	.004
	Sig. (2-tailed)	.173	.462	.529	.501	.915
	N	1105	1105	1118	989	747

Correlations

		MEDICAT	AGEWEAN	BRFEED	DUMMY	STILLDMY
TMEAL	Pearson Correlation	-.008	.029	-.002	.106**	.113**
	Sig. (2-tailed)	.793	.331	.942	.001	.002
	N	1104	1104	1117	988	747
SMEAL	Pearson Correlation	.033	.060*	-.114**	.084**	.091*
	Sig. (2-tailed)	.273	.045	.000	.009	.013
	N	1104	1104	1117	988	746
NMEAL	Pearson Correlation	-.008	.025	-.025	.021	.[a]
	Sig. (2-tailed)	.800	.408	.408	.500	.
	N	1106	1106	1119	990	748
BVESSEL	Pearson Correlation	.004	.066*	-.114**	.031	.017
	Sig. (2-tailed)	.904	.030	.000	.333	.651
	N	1094	1094	1106	980	739
FVESSEL	Pearson Correlation	-.026	-.075*	.057	-.015	.007
	Sig. (2-tailed)	.389	.014	.057	.634	.842
	N	1093	1093	1105	979	738
CVESSEL	Pearson Correlation	.004	.003	.013	-.048	.014
	Sig. (2-tailed)	.885	.909	.659	.134	.698
	N	1092	1092	1104	978	737
SNACK	Pearson Correlation	-.009	.083**	-.091**	.052	.025
	Sig. (2-tailed)	.782	.007	.003	.106	.500
	N	1064	1064	1076	952	724
BSNACK	Pearson Correlation	-.042	.042	-.022	.054	.025
	Sig. (2-tailed)	.180	.173	.472	.100	.514
	N	1037	1035	1047	925	704
SSNACK	Pearson Correlation	-.015	.046	-.039	.082*	.017
	Sig. (2-tailed)	.621	.139	.213	.012	.649
	N	1036	1034	1046	924	704
CSNACK	Pearson Correlation	.002	.109**	-.153**	.043	.063
	Sig. (2-tailed)	.939	.000	.000	.194	.096
	N	1037	1035	1047	925	704
FSNACK	Pearson Correlation	.025	-.013	.049	.020	-.027
	Sig. (2-tailed)	.427	.674	.111	.547	.474
	N	1035	1033	1045	924	703
TOOTHPAS	Pearson Correlation	-.036	-.007	.093**	-.052	-.063
	Sig. (2-tailed)	.256	.822	.003	.119	.100
	N	1018	1020	1032	910	689
FLUOR	Pearson Correlation	-.075*	-.026	-.033	-.034	.032
	Sig. (2-tailed)	.014	.384	.281	.286	.381
	N	1088	1087	1100	972	732
CARE	Pearson Correlation	.032	-.045	.038	.029	.010
	Sig. (2-tailed)	.292	.137	.212	.368	.785
	N	1089	1089	1102	975	737
SHOPTYPE	Pearson Correlation	.049	.078**	-.081**	.059	.031
	Sig. (2-tailed)	.102	.010	.007	.065	.391
	N	1103	1103	1115	989	746
BEDTIME	Pearson Correlation	.011	.070*	-.125**	.082*	.062
	Sig. (2-tailed)	.727	.020	.000	.010	.093
	N	1095	1095	1108	981	740

Correlations

		MEDICAT	AGEWEAN	BRFEED	DUMMY	STILLDMY
NIGHT	Pearson Correlation	.018	.034	.007	-.080*	.013
	Sig. (2-tailed)	.548	.264	.828	.013	.719
	N	1075	1076	1087	962	728
WHOBRUSH	Pearson Correlation	-.045	-.035	.124**	-.103**	-.089*
	Sig. (2-tailed)	.144	.248	.000	.002	.018
	N	1068	1069	1081	955	718

Correlations

		VITAMIN	SIBLINGS	MUM_AGE	MARSTAT	SC
DEPCAT	Pearson Correlation	.131**	-.011	-.313**	.371**	.427**
	Sig. (2-tailed)	.000	.698	.000	.000	.000
	N	1079	1163	1121	1060	930
SMM	Pearson Correlation	.008	.058	.023	.052	.028
	Sig. (2-tailed)	.819	.083	.492	.137	.451
	N	851	911	881	831	726
SSM	Pearson Correlation	.073*	-.015	-.016	.025	.017
	Sig. (2-tailed)	.034	.655	.636	.469	.640
	N	851	911	881	831	726
STM	Pearson Correlation	.017	.075*	.013	.055	.025
	Sig. (2-tailed)	.620	.023	.693	.116	.509
	N	851	911	881	831	726
LACTM	Pearson Correlation	.082*	.086**	.000	.087*	.092*
	Sig. (2-tailed)	.016	.008	.996	.011	.012
	N	874	933	903	852	745
YEASTM	Pearson Correlation	.048	.059	-.009	-.045	.005
	Sig. (2-tailed)	.165	.079	.796	.194	.889
	N	843	900	870	820	716
SM	Pearson Correlation	.017	.066*	.054	.039	.013
	Sig. (2-tailed)	.584	.028	.078	.216	.702
	N	1044	1121	1078	1018	897
SS	Pearson Correlation	.004	.012	.035	-.015	-.001
	Sig. (2-tailed)	.892	.680	.248	.639	.971
	N	1044	1121	1078	1018	897
ST	Pearson Correlation	.011	.071*	.073*	.031	.010
	Sig. (2-tailed)	.723	.018	.016	.325	.764
	N	1044	1121	1078	1018	897
LACT	Pearson Correlation	.004	.047	-.008	.024	.042
	Sig. (2-tailed)	.892	.110	.788	.447	.200
	N	1060	1138	1096	1035	912
YEAST	Pearson Correlation	.024	-.029	-.048	.058	-.011
	Sig. (2-tailed)	.437	.327	.117	.063	.741
	N	1034	1111	1069	1010	888
SEX	Pearson Correlation	.025	-.017	-.031	.010	-.023
	Sig. (2-tailed)	.408	.573	.292	.754	.486
	N	1079	1164	1122	1060	931
HIGHRISK	Pearson Correlation	-.171**	-.078*	.204**	-.376**	-.299**
	Sig. (2-tailed)	.000	.011	.000	.000	.000
	N	1001	1072	1034	983	862
WTCENT	Pearson Correlation	-.004	.069*	.051	-.021	-.009
	Sig. (2-tailed)	.903	.028	.109	.516	.804
	N	930	1008	978	917	813
HTCENT	Pearson Correlation	-.004	.048	-.002	-.021	.002
	Sig. (2-tailed)	.891	.124	.940	.529	.950
	N	933	1014	981	930	816
WTCENTA	Pearson Correlation	.076*	.056	.002	-.038	-.042
	Sig. (2-tailed)	.015	.062	.958	.222	.212
	N	1036	1120	1077	1019	897
HTCENTA	Pearson Correlation	.028	.008	.005	-.015	.004
	Sig. (2-tailed)	.368	.799	.882	.646	.896
	N	1017	1100	1058	1002	887

Correlations

		VITAMIN	SIBLINGS	MUM_AGE	MARSTAT	SC
WTCENTB	Pearson Correlation	.020	-.033	-.014	-.045	-.040
	Sig. (2-tailed)	.521	.277	.637	.151	.226
	N	1036	1116	1077	1015	902
HTCENTB	Pearson Correlation	.049	-.052	.016	-.020	-.097**
	Sig. (2-tailed)	.119	.086	.608	.526	.004
	N	1031	1111	1072	1011	900
CIMMUN	Pearson Correlation	.048	.092**	.023	.043	.004
	Sig. (2-tailed)	.119	.002	.454	.163	.901
	N	1048	1133	1092	1032	903
MEDICAT	Pearson Correlation	.038	-.053	.046	-.033	.016
	Sig. (2-tailed)	.222	.076	.124	.292	.634
	N	1058	1140	1098	1037	912
AGEWEAN	Pearson Correlation	-.139**	.025	.175**	-.168**	-.159**
	Sig. (2-tailed)	.000	.406	.000	.000	.000
	N	1061	1135	1096	1034	913
BRFEED	Pearson Correlation	.182**	-.003	-.230**	.307**	.327**
	Sig. (2-tailed)	.000	.926	.000	.000	.000
	N	1070	1151	1111	1048	920
DUMMY	Pearson Correlation	-.048	.095**	.146**	-.125**	-.201**
	Sig. (2-tailed)	.134	.003	.000	.000	.000
	N	970	1015	983	928	816
STILLDMY	Pearson Correlation	-.098**	.052	.067	-.114**	-.149**
	Sig. (2-tailed)	.007	.150	.069	.002	.000
	N	739	770	748	703	611
VITAMIN	Pearson Correlation	1.000	.125**	-.072*	.136**	.170**
	Sig. (2-tailed)	.	.000	.021	.000	.000
	N	1079	1066	1031	973	855
SIBLINGS	Pearson Correlation	.125**	1.000	.357**	-.106**	.002
	Sig. (2-tailed)	.000	.	.000	.001	.941
	N	1066	1164	1114	1051	920
MUM_AGE	Pearson Correlation	-.072*	.357**	1.000	-.404**	-.231**
	Sig. (2-tailed)	.021	.000	.	.000	.000
	N	1031	1114	1122	1017	890
MARSTAT	Pearson Correlation	.136**	-.106**	-.404**	1.000	.383**
	Sig. (2-tailed)	.000	.001	.000	.	.000
	N	973	1051	1017	1060	844
SC	Pearson Correlation	.170**	.002	-.231**	.383**	1.000
	Sig. (2-tailed)	.000	.941	.000	.000	.
	N	855	920	890	844	931
SMOKE	Pearson Correlation	-.164**	-.059	.147**	-.355**	-.289**
	Sig. (2-tailed)	.000	.064	.000	.000	.000
	N	936	1006	966	915	793
EMPLOY	Pearson Correlation	.098**	.042	-.298**	.424**	.187**
	Sig. (2-tailed)	.001	.154	.000	.000	.000
	N	1063	1146	1105	1049	924
BMEAL	Pearson Correlation	.005	.029	-.039	-.003	.023
	Sig. (2-tailed)	.868	.329	.200	.914	.492
	N	1039	1117	1077	1015	902
DMEAL	Pearson Correlation	.025	.000	-.083**	.125**	.099**
	Sig. (2-tailed)	.412	.997	.006	.000	.003
	N	1039	1117	1077	1015	901

Correlations

		VITAMIN	SIBLINGS	MUM_AGE	MARSTAT	SC
TMEAL	Pearson Correlation	-.049	.053	-.003	-.045	-.056
	Sig. (2-tailed)	.117	.078	.922	.150	.091
	N	1038	1116	1076	1014	900
SMEAL	Pearson Correlation	-.026	-.020	.068*	-.131**	-.066*
	Sig. (2-tailed)	.406	.501	.025	.000	.047
	N	1038	1116	1076	1014	901
NMEAL	Pearson Correlation	-.020	-.041	-.038	-.041	.[a]
	Sig. (2-tailed)	.518	.171	.214	.188	.
	N	1040	1118	1078	1016	902
BVESSEL	Pearson Correlation	-.059	-.056	.066*	-.136**	-.107**
	Sig. (2-tailed)	.057	.062	.033	.000	.001
	N	1028	1105	1065	1005	895
FVESSEL	Pearson Correlation	.046	.067*	-.079*	.131**	.145**
	Sig. (2-tailed)	.139	.026	.010	.000	.000
	N	1027	1104	1064	1004	895
CVESSEL	Pearson Correlation	.021	.059*	.042	-.036	-.034
	Sig. (2-tailed)	.496	.049	.175	.249	.312
	N	1026	1103	1063	1003	895
SNACK	Pearson Correlation	-.043	.008	.037	-.052	-.016
	Sig. (2-tailed)	.174	.795	.237	.105	.636
	N	1002	1075	1038	978	871
BSNACK	Pearson Correlation	-.074*	-.098**	-.073*	.037	-.025
	Sig. (2-tailed)	.022	.002	.020	.260	.476
	N	974	1046	1008	954	847
SSNACK	Pearson Correlation	-.090**	.019	.119**	-.093**	-.047
	Sig. (2-tailed)	.005	.539	.000	.004	.167
	N	973	1045	1007	953	847
CSNACK	Pearson Correlation	-.141**	-.046	.165**	-.249**	-.114**
	Sig. (2-tailed)	.000	.137	.000	.000	.001
	N	974	1046	1008	954	847
FSNACK	Pearson Correlation	.029	.022	.023	-.026	-.022
	Sig. (2-tailed)	.361	.474	.459	.426	.525
	N	972	1044	1006	952	847
TOOTHPAS	Pearson Correlation	.091**	.161**	.056	.068*	.094**
	Sig. (2-tailed)	.005	.000	.076	.038	.006
	N	960	1029	996	937	854
FLUOR	Pearson Correlation	-.105**	-.054	.003	-.045	-.087**
	Sig. (2-tailed)	.001	.075	.930	.154	.010
	N	1025	1099	1059	999	892
CARE	Pearson Correlation	.003	.045	.001	.019	.072*
	Sig. (2-tailed)	.913	.138	.969	.541	.031
	N	1023	1101	1062	1001	891
SHOPTYPE	Pearson Correlation	-.011	.065*	.140**	-.228**	-.156**
	Sig. (2-tailed)	.727	.031	.000	.000	.000
	N	1036	1114	1075	1013	899
BEDTIME	Pearson Correlation	-.031	-.001	.125**	-.155**	-.091**
	Sig. (2-tailed)	.321	.976	.000	.000	.007
	N	1030	1107	1067	1006	894

Correlations

		VITAMIN	SIBLINGS	MUM_AGE	MARSTAT	SC
NIGHT	Pearson Correlation	.009	-.034	.047	-.055	-.078*
	Sig. (2-tailed)	.785	.257	.126	.083	.021
	N	1008	1086	1046	991	879
WHOBRUSH	Pearson Correlation	.066*	.137**	.006	.120**	.118**
	Sig. (2-tailed)	.038	.000	.847	.000	.000
	N	1001	1079	1041	979	875

Correlations

		SMOKE	EMPLOY	BMEAL	DMEAL	TMEAL
DEPCAT	Pearson Correlation	-.251**	.278**	.021	.058*	.020
	Sig. (2-tailed)	.000	.000	.474	.048	.503
	N	1013	1158	1158	1158	1157
SMM	Pearson Correlation	-.086*	.005	.045	.033	-.011
	Sig. (2-tailed)	.016	.881	.175	.314	.737
	N	791	910	924	925	925
SSM	Pearson Correlation	-.068	.027	.004	-.016	-.030
	Sig. (2-tailed)	.054	.409	.913	.623	.364
	N	791	910	924	925	925
STM	Pearson Correlation	-.082*	.017	.042	.029	-.014
	Sig. (2-tailed)	.021	.601	.207	.378	.673
	N	791	910	924	925	925
LACTM	Pearson Correlation	-.170**	.126**	.071*	.011	-.048
	Sig. (2-tailed)	.000	.000	.030	.726	.139
	N	810	932	948	949	949
YEASTM	Pearson Correlation	-.047	.031	-.011	.045	-.038
	Sig. (2-tailed)	.185	.347	.730	.175	.248
	N	783	898	913	914	914
SM	Pearson Correlation	-.080*	.049	-.045	.061*	.040
	Sig. (2-tailed)	.012	.104	.134	.042	.175
	N	981	1116	1124	1124	1123
SS	Pearson Correlation	.031	-.029	-.014	-.016	-.012
	Sig. (2-tailed)	.324	.326	.648	.585	.696
	N	981	1116	1124	1124	1123
ST	Pearson Correlation	-.082**	.048	-.046	.058	.039
	Sig. (2-tailed)	.010	.112	.127	.052	.196
	N	981	1116	1124	1124	1123
LACT	Pearson Correlation	-.047	.054	.007	-.005	.017
	Sig. (2-tailed)	.142	.067	.817	.865	.568
	N	992	1134	1141	1141	1140
YEAST	Pearson Correlation	-.049	.069*	.003	-.034	-.012
	Sig. (2-tailed)	.131	.022	.913	.252	.693
	N	968	1109	1113	1113	1112
SEX	Pearson Correlation	.025	-.006	.028	.008	.047
	Sig. (2-tailed)	.428	.836	.348	.781	.112
	N	1014	1159	1133	1133	1132
HIGHRISK	Pearson Correlation	.308**	-.316**	-.089**	-.143**	.007
	Sig. (2-tailed)	.000	.000	.004	.000	.812
	N	948	1069	1057	1057	1056
WTCENT	Pearson Correlation	.064	-.001	-.047	-.055	-.045
	Sig. (2-tailed)	.059	.975	.140	.087	.158
	N	874	1004	983	983	982
HTCENT	Pearson Correlation	.045	-.013	-.014	.016	.056
	Sig. (2-tailed)	.181	.687	.670	.618	.078
	N	878	1009	985	986	985
WTCENTA	Pearson Correlation	.013	-.036	-.001	-.023	-.016
	Sig. (2-tailed)	.685	.228	.981	.443	.591
	N	975	1115	1090	1090	1089
HTCENTA	Pearson Correlation	.060	-.052	-.051	.000	.002
	Sig. (2-tailed)	.063	.087	.092	.995	.954
	N	954	1095	1072	1072	1071

Correlations

		SMOKE	EMPLOY	BMEAL	DMEAL	TMEAL
WTCENTB	Pearson Correlation	.039	-.066*	-.045	-.013	-.007
	Sig. (2-tailed)	.225	.029	.137	.672	.817
	N	977	1113	1089	1089	1088
HTCENTB	Pearson Correlation	.046	-.064*	-.032	.000	.041
	Sig. (2-tailed)	.152	.033	.299	.995	.177
	N	972	1108	1085	1085	1084
CIMMUN	Pearson Correlation	-.076*	.041	.011	.001	-.021
	Sig. (2-tailed)	.017	.169	.722	.975	.493
	N	986	1126	1099	1099	1098
MEDICAT	Pearson Correlation	-.015	-.033	-.025	-.041	-.008
	Sig. (2-tailed)	.647	.261	.411	.173	.793
	N	992	1133	1105	1105	1104
AGEWEAN	Pearson Correlation	.213**	-.097**	.046	-.022	.029
	Sig. (2-tailed)	.000	.001	.125	.462	.331
	N	991	1131	1105	1105	1104
BRFEED	Pearson Correlation	-.244**	.218**	.038	.019	-.002
	Sig. (2-tailed)	.000	.000	.198	.529	.942
	N	1004	1146	1118	1118	1117
DUMMY	Pearson Correlation	.152**	-.105**	.001	.021	.106**
	Sig. (2-tailed)	.000	.001	.982	.501	.001
	N	888	1014	989	989	988
STILLDMY	Pearson Correlation	.203**	-.078*	.017	.004	.113**
	Sig. (2-tailed)	.000	.031	.635	.915	.002
	N	674	765	748	747	747
VITAMIN	Pearson Correlation	-.164**	.098**	.005	.025	-.049
	Sig. (2-tailed)	.000	.001	.868	.412	.117
	N	936	1063	1039	1039	1038
SIBLINGS	Pearson Correlation	-.059	.042	.029	.000	.053
	Sig. (2-tailed)	.064	.154	.329	.997	.078
	N	1006	1146	1117	1117	1116
MUM_AGE	Pearson Correlation	.147**	-.298**	-.039	-.083**	-.003
	Sig. (2-tailed)	.000	.000	.200	.006	.922
	N	966	1105	1077	1077	1076
MARSTAT	Pearson Correlation	-.355**	.424**	-.003	.125**	-.045
	Sig. (2-tailed)	.000	.000	.914	.000	.150
	N	915	1049	1015	1015	1014
SC	Pearson Correlation	-.289**	.187**	.023	.099**	-.056
	Sig. (2-tailed)	.000	.000	.492	.003	.091
	N	793	924	902	901	900
SMOKE	Pearson Correlation	1.000	-.248**	-.039	-.118**	.068*
	Sig. (2-tailed)	.	.000	.228	.000	.034
	N	1014	1000	976	976	975
EMPLOY	Pearson Correlation	-.248**	1.000	.048	.080**	-.014
	Sig. (2-tailed)	.000	.	.113	.008	.641
	N	1000	1159	1113	1113	1112
BMEAL	Pearson Correlation	-.039	.048	1.000	.024	-.014
	Sig. (2-tailed)	.228	.113	.	.416	.627
	N	976	1113	1159	1158	1157
DMEAL	Pearson Correlation	-.118**	.080**	.024	1.000	-.017
	Sig. (2-tailed)	.000	.008	.416	.	.554
	N	976	1113	1158	1159	1158

Correlations

		SMOKE	EMPLOY	BMEAL	DMEAL	TMEAL
TMEAL	Pearson Correlation	.068*	-.014	-.014	-.017	1.000
	Sig. (2-tailed)	.034	.641	.627	.554	.
	N	975	1112	1157	1158	1158
SMEAL	Pearson Correlation	.136**	-.145**	.003	-.027	-.067*
	Sig. (2-tailed)	.000	.000	.928	.351	.023
	N	975	1112	1155	1156	1155
NMEAL	Pearson Correlation	.054	-.062*	.[a]	.005	.003
	Sig. (2-tailed)	.092	.040	.	.875	.912
	N	977	1114	1156	1156	1155
BVESSEL	Pearson Correlation	.143**	-.115**	-.052	-.071*	.007
	Sig. (2-tailed)	.000	.000	.079	.017	.806
	N	967	1101	1146	1146	1145
FVESSEL	Pearson Correlation	-.129**	.087**	-.012	.096**	.006
	Sig. (2-tailed)	.000	.004	.692	.001	.836
	N	966	1100	1144	1144	1143
CVESSEL	Pearson Correlation	-.016	-.031	.014	-.037	-.022
	Sig. (2-tailed)	.629	.307	.630	.217	.452
	N	965	1099	1144	1144	1143
SNACK	Pearson Correlation	.127**	-.056	-.050	-.011	-.022
	Sig. (2-tailed)	.000	.067	.095	.716	.457
	N	947	1071	1115	1115	1114
BSNACK	Pearson Correlation	.022	-.009	-.002	.000	-.015
	Sig. (2-tailed)	.507	.760	.939	.989	.626
	N	920	1042	1086	1086	1085
SSNACK	Pearson Correlation	.092**	-.070*	-.023	-.027	-.015
	Sig. (2-tailed)	.005	.025	.446	.376	.633
	N	919	1041	1085	1085	1084
CSNACK	Pearson Correlation	.179**	-.111**	.035	-.067*	-.014
	Sig. (2-tailed)	.000	.000	.246	.027	.646
	N	920	1042	1086	1086	1085
FSNACK	Pearson Correlation	-.055	.011	-.017	-.011	.002
	Sig. (2-tailed)	.098	.721	.585	.728	.937
	N	918	1040	1084	1084	1083
TOOTHPAS	Pearson Correlation	-.066*	.099**	-.026	.028	.065*
	Sig. (2-tailed)	.048	.002	.391	.353	.035
	N	897	1026	1065	1065	1064
FLUOR	Pearson Correlation	.037	-.013	-.007	-.017	.004
	Sig. (2-tailed)	.250	.674	.802	.557	.885
	N	960	1095	1136	1136	1135
CARE	Pearson Correlation	-.038	.038	-.002	.035	-.047
	Sig. (2-tailed)	.242	.209	.958	.237	.110
	N	961	1097	1139	1139	1138
SHOPTYPE	Pearson Correlation	.118**	-.254**	-.004	-.070*	.020
	Sig. (2-tailed)	.000	.000	.889	.018	.491
	N	974	1110	1154	1154	1153
BEDTIME	Pearson Correlation	.144**	-.159**	.026	-.111**	.075*
	Sig. (2-tailed)	.000	.000	.387	.000	.012
	N	970	1103	1146	1146	1145

Correlations

		SMOKE	EMPLOY	BMEAL	DMEAL	TMEAL
NIGHT	Pearson Correlation	.058	-.051	-.012	-.046	.008
	Sig. (2-tailed)	.076	.091	.692	.121	.794
	N	952	1082	1125	1125	1124
WHOBRUSH	Pearson Correlation	-.117**	.155**	.005	.067*	.069*
	Sig. (2-tailed)	.000	.000	.865	.025	.021
	N	945	1075	1119	1119	1118

Correlations

		SMEAL	NMEAL	BVESSEL	FVESSEL	CVESSEL
DEPCAT	Pearson Correlation	-.125**	.006	-.143**	.113**	-.078**
	Sig. (2-tailed)	.000	.842	.000	.000	.008
	N	1155	1156	1146	1144	1144
SMM	Pearson Correlation	-.019	.033	.001	.061	-.063
	Sig. (2-tailed)	.569	.314	.974	.064	.059
	N	923	922	914	912	913
SSM	Pearson Correlation	.009	.009	-.059	.032	.022
	Sig. (2-tailed)	.788	.788	.077	.341	.501
	N	923	922	914	912	913
STM	Pearson Correlation	-.011	.034	-.012	.062	-.055
	Sig. (2-tailed)	.731	.302	.708	.059	.096
	N	923	922	914	912	913
LACTM	Pearson Correlation	-.070*	-.040	-.047	.021	-.005
	Sig. (2-tailed)	.031	.218	.146	.514	.879
	N	947	946	938	936	937
YEASTM	Pearson Correlation	-.010	-.095**	-.005	.056	-.009
	Sig. (2-tailed)	.768	.004	.877	.093	.795
	N	912	911	903	901	902
SM	Pearson Correlation	-.040	.010	-.103**	.051	.024
	Sig. (2-tailed)	.179	.736	.001	.088	.424
	N	1122	1122	1112	1110	1111
SS	Pearson Correlation	.014	.003	.009	-.002	.002
	Sig. (2-tailed)	.639	.917	.774	.940	.952
	N	1122	1122	1112	1110	1111
ST	Pearson Correlation	-.028	.010	-.091**	.044	.027
	Sig. (2-tailed)	.342	.731	.003	.147	.364
	N	1122	1122	1112	1110	1111
LACT	Pearson Correlation	-.034	.006	.002	.032	.016
	Sig. (2-tailed)	.249	.834	.955	.290	.595
	N	1139	1139	1129	1127	1128
YEAST	Pearson Correlation	.021	-.088**	-.051	.069*	.001
	Sig. (2-tailed)	.493	.003	.092	.021	.985
	N	1111	1111	1101	1099	1100
SEX	Pearson Correlation	-.002	-.031	.044	-.019	-.034
	Sig. (2-tailed)	.955	.291	.140	.528	.252
	N	1132	1134	1121	1120	1119
HIGHRISK	Pearson Correlation	.047	.052	.198**	-.189**	.061*
	Sig. (2-tailed)	.126	.091	.000	.000	.049
	N	1056	1057	1046	1045	1044
WTCENT	Pearson Correlation	.051	.[a]	.060	-.054	.021
	Sig. (2-tailed)	.108	.	.063	.092	.506
	N	982	983	972	971	970
HTCENT	Pearson Correlation	.055	.057	.077*	.004	-.032
	Sig. (2-tailed)	.087	.072	.016	.903	.317
	N	985	986	974	973	972
WTCENTA	Pearson Correlation	.004	.066*	.062*	-.035	-.067*
	Sig. (2-tailed)	.883	.030	.041	.256	.028
	N	1089	1091	1078	1077	1076
HTCENTA	Pearson Correlation	.048	.070*	.039	.014	-.060
	Sig. (2-tailed)	.116	.022	.203	.647	.050
	N	1071	1073	1060	1059	1058

Correlations

		SMEAL	NMEAL	BVESSEL	FVESSEL	CVESSEL
WTCENTB	Pearson Correlation	.018	-.012	.025	-.022	-.009
	Sig. (2-tailed)	.552	.682	.415	.464	.763
	N	1088	1090	1078	1077	1076
HTCENTB	Pearson Correlation	.011	-.012	.046	-.030	-.026
	Sig. (2-tailed)	.717	.698	.136	.329	.389
	N	1084	1086	1074	1073	1072
CIMMUN	Pearson Correlation	-.027	.006	-.020	.057	-.060*
	Sig. (2-tailed)	.367	.844	.510	.060	.050
	N	1098	1100	1087	1086	1085
MEDICAT	Pearson Correlation	.033	-.008	.004	-.026	.004
	Sig. (2-tailed)	.273	.800	.904	.389	.885
	N	1104	1106	1094	1093	1092
AGEWEAN	Pearson Correlation	.060*	.025	.066*	-.075*	.003
	Sig. (2-tailed)	.045	.408	.030	.014	.909
	N	1104	1106	1094	1093	1092
BRFEED	Pearson Correlation	-.114**	-.025	-.114**	.057	.013
	Sig. (2-tailed)	.000	.408	.000	.057	.659
	N	1117	1119	1106	1105	1104
DUMMY	Pearson Correlation	.084**	.021	.031	-.015	-.048
	Sig. (2-tailed)	.009	.500	.333	.634	.134
	N	988	990	980	979	978
STILLDMY	Pearson Correlation	.091*	.[a]	.017	.007	.014
	Sig. (2-tailed)	.013	.	.651	.842	.698
	N	746	748	739	738	737
VITAMIN	Pearson Correlation	-.026	-.020	-.059	.046	.021
	Sig. (2-tailed)	.406	.518	.057	.139	.496
	N	1038	1040	1028	1027	1026
SIBLINGS	Pearson Correlation	-.020	-.041	-.056	.067*	.059*
	Sig. (2-tailed)	.501	.171	.062	.026	.049
	N	1116	1118	1105	1104	1103
MUM_AGE	Pearson Correlation	.068*	-.038	.066*	-.079*	.042
	Sig. (2-tailed)	.025	.214	.033	.010	.175
	N	1076	1078	1065	1064	1063
MARSTAT	Pearson Correlation	-.131**	-.041	-.136**	.131**	-.036
	Sig. (2-tailed)	.000	.188	.000	.000	.249
	N	1014	1016	1005	1004	1003
SC	Pearson Correlation	-.066*	.[a]	-.107**	.145**	-.034
	Sig. (2-tailed)	.047	.	.001	.000	.312
	N	901	902	895	895	895
SMOKE	Pearson Correlation	.136**	.054	.143**	-.129**	-.016
	Sig. (2-tailed)	.000	.092	.000	.000	.629
	N	975	977	967	966	965
EMPLOY	Pearson Correlation	-.145**	-.062*	-.115**	.087**	-.031
	Sig. (2-tailed)	.000	.040	.000	.004	.307
	N	1112	1114	1101	1100	1099
BMEAL	Pearson Correlation	.003	.[a]	-.052	-.012	.014
	Sig. (2-tailed)	.928	.	.079	.692	.630
	N	1155	1156	1146	1144	1144
DMEAL	Pearson Correlation	-.027	.005	-.071*	.096**	-.037
	Sig. (2-tailed)	.351	.875	.017	.001	.217
	N	1156	1156	1146	1144	1144

Correlations

		SMEAL	NMEAL	BVESSEL	FVESSEL	CVESSEL
TMEAL	Pearson Correlation	-.067*	.003	.007	.006	-.022
	Sig. (2-tailed)	.023	.912	.806	.836	.452
	N	1155	1155	1145	1143	1143
SMEAL	Pearson Correlation	1.000	.030	-.011	.028	.026
	Sig. (2-tailed)	.	.302	.720	.352	.383
	N	1156	1154	1143	1141	1142
NMEAL	Pearson Correlation	.030	1.000	-.027	.018	-.009
	Sig. (2-tailed)	.302	.	.356	.551	.758
	N	1154	1157	1144	1143	1142
BVESSEL	Pearson Correlation	-.011	-.027	1.000	-.448**	-.056
	Sig. (2-tailed)	.720	.356	.	.000	.058
	N	1143	1144	1147	1145	1145
FVESSEL	Pearson Correlation	.028	.018	-.448**	1.000	-.367**
	Sig. (2-tailed)	.352	.551	.000	.	.000
	N	1141	1143	1145	1145	1144
CVESSEL	Pearson Correlation	.026	-.009	-.056	-.367**	1.000
	Sig. (2-tailed)	.383	.758	.058	.000	.
	N	1142	1142	1145	1144	1145
SNACK	Pearson Correlation	.044	-.075*	-.071*	.057	.031
	Sig. (2-tailed)	.143	.013	.018	.057	.301
	N	1112	1113	1112	1110	1110
BSNACK	Pearson Correlation	-.017	-.036	-.094**	.080**	-.083**
	Sig. (2-tailed)	.578	.240	.002	.008	.007
	N	1083	1085	1083	1082	1081
SSNACK	Pearson Correlation	.017	-.014	.037	-.008	.013
	Sig. (2-tailed)	.568	.655	.219	.798	.680
	N	1082	1084	1082	1081	1080
CSNACK	Pearson Correlation	.076*	-.023	.085**	-.083**	.077*
	Sig. (2-tailed)	.013	.445	.005	.006	.011
	N	1083	1085	1083	1082	1081
FSNACK	Pearson Correlation	.051	.026	-.107**	.082**	.093**
	Sig. (2-tailed)	.096	.397	.000	.007	.002
	N	1081	1083	1081	1080	1079
TOOTHPAS	Pearson Correlation	.004	.016	-.115**	.130**	-.023
	Sig. (2-tailed)	.887	.591	.000	.000	.451
	N	1062	1063	1056	1054	1054
FLUOR	Pearson Correlation	-.027	.008	.061*	-.030	.019
	Sig. (2-tailed)	.369	.789	.040	.315	.518
	N	1134	1135	1124	1123	1123
CARE	Pearson Correlation	.010	-.007	-.019	.019	-.072*
	Sig. (2-tailed)	.734	.818	.513	.518	.016
	N	1136	1137	1127	1125	1125
SHOPTYPE	Pearson Correlation	-.028	-.047	.020	-.021	.049
	Sig. (2-tailed)	.349	.108	.491	.474	.099
	N	1151	1152	1143	1141	1141
BEDTIME	Pearson Correlation	.467**	.050	.048	-.006	.013
	Sig. (2-tailed)	.000	.091	.107	.829	.673
	N	1144	1145	1134	1132	1132

Correlations

		SMEAL	NMEAL	BVESSEL	FVESSEL	CVESSEL
NIGHT	Pearson Correlation	.033	-.016	.127**	-.097**	.022
	Sig. (2-tailed)	.270	.602	.000	.001	.455
	N	1122	1124	1115	1113	1113
WHOBRUSH	Pearson Correlation	-.011	.018	-.138**	.111**	-.005
	Sig. (2-tailed)	.716	.547	.000	.000	.867
	N	1116	1117	1107	1105	1105

Correlations

		SNACK	BSNACK	SSNACK	CSNACK	FSNACK
DEPCAT	Pearson Correlation	-.026	.063*	-.097**	-.187**	-.033
	Sig. (2-tailed)	.390	.039	.001	.000	.279
	N	1116	1086	1085	1086	1084
SMM	Pearson Correlation	.025	-.007	-.045	.044	.001
	Sig. (2-tailed)	.463	.827	.188	.202	.967
	N	888	861	860	861	860
SSM	Pearson Correlation	.004	.008	.023	-.018	-.035
	Sig. (2-tailed)	.894	.808	.503	.596	.303
	N	888	861	860	861	860
STM	Pearson Correlation	.015	-.006	-.034	.049	.004
	Sig. (2-tailed)	.666	.854	.322	.150	.915
	N	888	861	860	861	860
LACTM	Pearson Correlation	-.044	-.066*	.011	-.001	-.002
	Sig. (2-tailed)	.183	.048	.745	.969	.945
	N	911	885	884	885	884
YEASTM	Pearson Correlation	-.009	-.024	-.022	-.013	-.015
	Sig. (2-tailed)	.780	.477	.521	.698	.658
	N	876	851	850	851	850
SM	Pearson Correlation	-.002	.007	.001	-.040	.060
	Sig. (2-tailed)	.957	.818	.985	.193	.053
	N	1082	1053	1052	1053	1051
SS	Pearson Correlation	-.011	-.037	-.024	-.011	.000
	Sig. (2-tailed)	.717	.231	.441	.712	.995
	N	1082	1053	1052	1053	1051
ST	Pearson Correlation	.003	-.009	.006	-.030	.073*
	Sig. (2-tailed)	.912	.762	.846	.325	.018
	N	1082	1053	1052	1053	1051
LACT	Pearson Correlation	-.032	-.008	-.038	-.045	-.006
	Sig. (2-tailed)	.289	.803	.213	.140	.845
	N	1099	1069	1068	1069	1067
YEAST	Pearson Correlation	.005	.038	-.068*	-.007	.018
	Sig. (2-tailed)	.863	.221	.029	.819	.568
	N	1071	1042	1041	1042	1040
SEX	Pearson Correlation	-.040	.022	.004	-.001	-.068*
	Sig. (2-tailed)	.188	.478	.902	.982	.027
	N	1091	1062	1061	1062	1060
HIGHRISK	Pearson Correlation	.077*	.047	.141**	.186**	-.082**
	Sig. (2-tailed)	.013	.140	.000	.000	.009
	N	1020	994	993	994	993
WTCENT	Pearson Correlation	-.012	-.040	-.036	-.012	.002
	Sig. (2-tailed)	.722	.220	.274	.706	.953
	N	948	921	920	921	920
HTCENT	Pearson Correlation	.061	.036	-.008	.010	-.027
	Sig. (2-tailed)	.060	.277	.819	.764	.417
	N	951	924	923	924	923
WTCENTA	Pearson Correlation	-.028	-.003	-.008	.032	-.002
	Sig. (2-tailed)	.365	.930	.802	.300	.957
	N	1049	1022	1021	1022	1021
HTCENTA	Pearson Correlation	-.022	.017	-.039	.022	-.025
	Sig. (2-tailed)	.478	.590	.219	.480	.432
	N	1031	1005	1004	1005	1004

Correlations

		SNACK	BSNACK	SSNACK	CSNACK	FSNACK
WTCENTB	Pearson Correlation	.035	.012	-.009	.004	-.063*
	Sig. (2-tailed)	.263	.703	.769	.909	.043
	N	1049	1022	1021	1022	1021
HTCENTB	Pearson Correlation	-.028	-.003	-.037	-.009	-.090**
	Sig. (2-tailed)	.361	.916	.241	.777	.004
	N	1046	1018	1017	1018	1017
CIMMUN	Pearson Correlation	-.024	-.018	-.060	-.024	.023
	Sig. (2-tailed)	.440	.557	.056	.435	.463
	N	1058	1031	1030	1031	1029
MEDICAT	Pearson Correlation	-.009	-.042	-.015	.002	.025
	Sig. (2-tailed)	.782	.180	.621	.939	.427
	N	1064	1037	1036	1037	1035
AGEWEAN	Pearson Correlation	.083**	.042	.046	.109**	-.013
	Sig. (2-tailed)	.007	.173	.139	.000	.674
	N	1064	1035	1034	1035	1033
BRFEED	Pearson Correlation	-.091**	-.022	-.039	-.153**	.049
	Sig. (2-tailed)	.003	.472	.213	.000	.111
	N	1076	1047	1046	1047	1045
DUMMY	Pearson Correlation	.052	.054	.082*	.043	.020
	Sig. (2-tailed)	.106	.100	.012	.194	.547
	N	952	925	924	925	924
STILLDMY	Pearson Correlation	.025	.025	.017	.063	-.027
	Sig. (2-tailed)	.500	.514	.649	.096	.474
	N	724	704	704	704	703
VITAMIN	Pearson Correlation	-.043	-.074*	-.090**	-.141**	.029
	Sig. (2-tailed)	.174	.022	.005	.000	.361
	N	1002	974	973	974	972
SIBLINGS	Pearson Correlation	.008	-.098**	.019	-.046	.022
	Sig. (2-tailed)	.795	.002	.539	.137	.474
	N	1075	1046	1045	1046	1044
MUM_AGE	Pearson Correlation	.037	-.073*	.119**	.165**	.023
	Sig. (2-tailed)	.237	.020	.000	.000	.459
	N	1038	1008	1007	1008	1006
MARSTAT	Pearson Correlation	-.052	.037	-.093**	-.249**	-.026
	Sig. (2-tailed)	.105	.260	.004	.000	.426
	N	978	954	953	954	952
SC	Pearson Correlation	-.016	-.025	-.047	-.114**	-.022
	Sig. (2-tailed)	.636	.476	.167	.001	.525
	N	871	847	847	847	847
SMOKE	Pearson Correlation	.127**	.022	.092**	.179**	-.055
	Sig. (2-tailed)	.000	.507	.005	.000	.098
	N	947	920	919	920	918
EMPLOY	Pearson Correlation	-.056	-.009	-.070*	-.111**	.011
	Sig. (2-tailed)	.067	.760	.025	.000	.721
	N	1071	1042	1041	1042	1040
BMEAL	Pearson Correlation	-.050	-.002	-.023	.035	-.017
	Sig. (2-tailed)	.095	.939	.446	.246	.585
	N	1115	1086	1085	1086	1084
DMEAL	Pearson Correlation	-.011	.000	-.027	-.067*	-.011
	Sig. (2-tailed)	.716	.989	.376	.027	.728
	N	1115	1086	1085	1086	1084

Correlations

		SNACK	BSNACK	SSNACK	CSNACK	FSNACK
TMEAL	Pearson Correlation	-.022	-.015	-.015	-.014	.002
	Sig. (2-tailed)	.457	.626	.633	.646	.937
	N	1114	1085	1084	1085	1083
SMEAL	Pearson Correlation	.044	-.017	.017	.076*	.051
	Sig. (2-tailed)	.143	.578	.568	.013	.096
	N	1112	1083	1082	1083	1081
NMEAL	Pearson Correlation	-.075*	-.036	-.014	-.023	.026
	Sig. (2-tailed)	.013	.240	.655	.445	.397
	N	1113	1085	1084	1085	1083
BVESSEL	Pearson Correlation	-.071*	-.094**	.037	.085**	-.107**
	Sig. (2-tailed)	.018	.002	.219	.005	.000
	N	1112	1083	1082	1083	1081
FVESSEL	Pearson Correlation	.057	.080**	-.008	-.083**	.082**
	Sig. (2-tailed)	.057	.008	.798	.006	.007
	N	1110	1082	1081	1082	1080
CVESSEL	Pearson Correlation	.031	-.083**	.013	.077*	.093**
	Sig. (2-tailed)	.301	.007	.680	.011	.002
	N	1110	1081	1080	1081	1079
SNACK	Pearson Correlation	1.000	.386**	.122**	.245**	.326**
	Sig. (2-tailed)	.	.000	.000	.000	.000
	N	1117	1080	1079	1080	1078
BSNACK	Pearson Correlation	.386**	1.000	.052	-.010	.029
	Sig. (2-tailed)	.000	.	.089	.739	.333
	N	1080	1087	1086	1087	1085
SSNACK	Pearson Correlation	.122**	.052	1.000	.174**	.091**
	Sig. (2-tailed)	.000	.089	.	.000	.003
	N	1079	1086	1086	1086	1085
CSNACK	Pearson Correlation	.245**	-.010	.174**	1.000	.126**
	Sig. (2-tailed)	.000	.739	.000	.	.000
	N	1080	1087	1086	1087	1085
FSNACK	Pearson Correlation	.326**	.029	.091**	.126**	1.000
	Sig. (2-tailed)	.000	.333	.003	.000	.
	N	1078	1085	1085	1085	1085
TOOTHPAS	Pearson Correlation	.045	.017	-.044	-.033	.117**
	Sig. (2-tailed)	.145	.594	.163	.289	.000
	N	1035	1014	1014	1014	1014
FLUOR	Pearson Correlation	.008	.008	.028	-.030	-.018
	Sig. (2-tailed)	.803	.786	.366	.322	.558
	N	1094	1066	1065	1066	1064
CARE	Pearson Correlation	-.012	-.014	-.021	-.006	-.008
	Sig. (2-tailed)	.694	.646	.490	.835	.794
	N	1098	1070	1069	1070	1068
SHOPTYPE	Pearson Correlation	.084**	.017	-.005	.129**	-.031
	Sig. (2-tailed)	.005	.567	.881	.000	.305
	N	1112	1084	1083	1084	1082
BEDTIME	Pearson Correlation	.072*	.053	.052	.087**	.042
	Sig. (2-tailed)	.017	.082	.090	.004	.168
	N	1104	1074	1073	1074	1072

Correlations

		SNACK	BSNACK	SSNACK	CSNACK	FSNACK
NIGHT	Pearson Correlation	-.048	-.061*	-.030	.066*	-.025
	Sig. (2-tailed)	.110	.046	.325	.031	.420
	N	1086	1056	1055	1056	1055
WHOBRUSH	Pearson Correlation	.029	.000	-.108**	-.086**	.101**
	Sig. (2-tailed)	.343	.988	.000	.005	.001
	N	1080	1051	1051	1051	1050

Correlations

		TOOTHPAS	FLUOR	CARE	SHOPTYPE	BEDTIME
DEPCAT	Pearson Correlation	.058	-.003	-.006	-.166**	-.155**
	Sig. (2-tailed)	.061	.910	.848	.000	.000
	N	1065	1137	1140	1155	1147
SMM	Pearson Correlation	.029	-.068*	.063	-.054	-.023
	Sig. (2-tailed)	.393	.039	.059	.099	.490
	N	848	905	910	921	915
SSM	Pearson Correlation	.020	-.042	.024	.010	.010
	Sig. (2-tailed)	.567	.211	.465	.755	.756
	N	848	905	910	921	915
STM	Pearson Correlation	.028	-.067*	.059	-.051	-.023
	Sig. (2-tailed)	.418	.044	.076	.124	.484
	N	848	905	910	921	915
LACTM	Pearson Correlation	.028	-.036	.041	-.047	-.081*
	Sig. (2-tailed)	.412	.275	.205	.147	.013
	N	872	929	933	945	939
YEASTM	Pearson Correlation	-.021	.014	.033	.056	-.057
	Sig. (2-tailed)	.543	.683	.318	.093	.086
	N	839	894	899	910	904
SM	Pearson Correlation	.004	-.078**	.037	-.037	-.061*
	Sig. (2-tailed)	.897	.009	.218	.210	.042
	N	1032	1105	1106	1121	1113
SS	Pearson Correlation	.027	-.028	.024	.015	.056
	Sig. (2-tailed)	.386	.351	.422	.613	.062
	N	1032	1105	1106	1121	1113
ST	Pearson Correlation	.019	-.080**	.039	-.044	-.055
	Sig. (2-tailed)	.532	.007	.193	.144	.067
	N	1032	1105	1106	1121	1113
LACT	Pearson Correlation	.083**	-.004	.048	.019	-.009
	Sig. (2-tailed)	.007	.901	.105	.517	.754
	N	1048	1121	1123	1138	1130
YEAST	Pearson Correlation	-.008	-.045	.025	-.014	.013
	Sig. (2-tailed)	.791	.138	.400	.647	.671
	N	1021	1094	1095	1110	1103
SEX	Pearson Correlation	.005	-.007	-.043	.006	.003
	Sig. (2-tailed)	.872	.826	.154	.842	.917
	N	1045	1115	1117	1130	1123
HIGHRISK	Pearson Correlation	-.158**	.084**	-.045	.123**	.116**
	Sig. (2-tailed)	.000	.006	.151	.000	.000
	N	973	1042	1042	1055	1048
WTCENT	Pearson Correlation	-.035	.016	.006	.004	.059
	Sig. (2-tailed)	.288	.614	.859	.905	.064
	N	912	964	968	978	971
HTCENT	Pearson Correlation	-.050	-.001	-.010	-.027	.067*
	Sig. (2-tailed)	.130	.974	.761	.394	.036
	N	909	968	971	983	976
WTCENTA	Pearson Correlation	-.102**	.028	.030	.007	.075*
	Sig. (2-tailed)	.001	.355	.331	.827	.014
	N	1007	1072	1075	1087	1080
HTCENTA	Pearson Correlation	-.114**	-.008	.030	-.056	.078*
	Sig. (2-tailed)	.000	.798	.325	.067	.011
	N	992	1056	1059	1069	1062

Correlations

		TOOTHPAS	FLUOR	CARE	SHOPTYPE	BEDTIME
WTCENTB	Pearson Correlation	-.091**	-.002	.027	.041	.075*
	Sig. (2-tailed)	.004	.955	.370	.178	.013
	N	1004	1071	1074	1087	1079
HTCENTB	Pearson Correlation	-.105**	-.038	-.026	.005	.024
	Sig. (2-tailed)	.001	.220	.399	.859	.431
	N	1001	1067	1070	1082	1076
CIMMUN	Pearson Correlation	.018	.008	-.021	-.045	-.003
	Sig. (2-tailed)	.569	.783	.485	.138	.915
	N	1012	1081	1083	1097	1089
MEDICAT	Pearson Correlation	-.036	-.075*	.032	.049	.011
	Sig. (2-tailed)	.256	.014	.292	.102	.727
	N	1018	1088	1089	1103	1095
AGEWEAN	Pearson Correlation	-.007	-.026	-.045	.078**	.070*
	Sig. (2-tailed)	.822	.384	.137	.010	.020
	N	1020	1087	1089	1103	1095
BRFEED	Pearson Correlation	.093**	-.033	.038	-.081**	-.125**
	Sig. (2-tailed)	.003	.281	.212	.007	.000
	N	1032	1100	1102	1115	1108
DUMMY	Pearson Correlation	-.052	-.034	.029	.059	.082*
	Sig. (2-tailed)	.119	.286	.368	.065	.010
	N	910	972	975	989	981
STILLDMY	Pearson Correlation	-.063	.032	.010	.031	.062
	Sig. (2-tailed)	.100	.381	.785	.391	.093
	N	689	732	737	746	740
VITAMIN	Pearson Correlation	.091**	-.105**	.003	-.011	-.031
	Sig. (2-tailed)	.005	.001	.913	.727	.321
	N	960	1025	1023	1036	1030
SIBLINGS	Pearson Correlation	.161**	-.054	.045	.065*	-.001
	Sig. (2-tailed)	.000	.075	.138	.031	.976
	N	1029	1099	1101	1114	1107
MUM_AGE	Pearson Correlation	.056	.003	.001	.140**	.125**
	Sig. (2-tailed)	.076	.930	.969	.000	.000
	N	996	1059	1062	1075	1067
MARSTAT	Pearson Correlation	.068*	-.045	.019	-.228**	-.155**
	Sig. (2-tailed)	.038	.154	.541	.000	.000
	N	937	999	1001	1013	1006
SC	Pearson Correlation	.094**	-.087**	.072*	-.156**	-.091**
	Sig. (2-tailed)	.006	.010	.031	.000	.007
	N	854	892	891	899	894
SMOKE	Pearson Correlation	-.066*	.037	-.038	.118**	.144**
	Sig. (2-tailed)	.048	.250	.242	.000	.000
	N	897	960	961	974	970
EMPLOY	Pearson Correlation	.099**	-.013	.038	-.254**	-.159**
	Sig. (2-tailed)	.002	.674	.209	.000	.000
	N	1026	1095	1097	1110	1103
BMEAL	Pearson Correlation	-.026	-.007	-.002	-.004	.026
	Sig. (2-tailed)	.391	.802	.958	.889	.387
	N	1065	1136	1139	1154	1146
DMEAL	Pearson Correlation	.028	-.017	.035	-.070*	-.111**
	Sig. (2-tailed)	.353	.557	.237	.018	.000
	N	1065	1136	1139	1154	1146

Correlations

		TOOTHPAS	FLUOR	CARE	SHOPTYPE	BEDTIME
TMEAL	Pearson Correlation	.065*	.004	-.047	.020	.075*
	Sig. (2-tailed)	.035	.885	.110	.491	.012
	N	1064	1135	1138	1153	1145
SMEAL	Pearson Correlation	.004	-.027	.010	-.028	.467**
	Sig. (2-tailed)	.887	.369	.734	.349	.000
	N	1062	1134	1136	1151	1144
NMEAL	Pearson Correlation	.016	.008	-.007	-.047	.050
	Sig. (2-tailed)	.591	.789	.818	.108	.091
	N	1063	1135	1137	1152	1145
BVESSEL	Pearson Correlation	-.115**	.061*	-.019	.020	.048
	Sig. (2-tailed)	.000	.040	.513	.491	.107
	N	1056	1124	1127	1143	1134
FVESSEL	Pearson Correlation	.130**	-.030	.019	-.021	-.006
	Sig. (2-tailed)	.000	.315	.518	.474	.829
	N	1054	1123	1125	1141	1132
CVESSEL	Pearson Correlation	-.023	.019	-.072*	.049	.013
	Sig. (2-tailed)	.451	.518	.016	.099	.673
	N	1054	1123	1125	1141	1132
SNACK	Pearson Correlation	.045	.008	-.012	.084**	.072*
	Sig. (2-tailed)	.145	.803	.694	.005	.017
	N	1035	1094	1098	1112	1104
BSNACK	Pearson Correlation	..017	.008	-.014	.017	.053
	Sig. (2-tailed)	.594	.786	.646	.567	.082
	N	1014	1066	1070	1084	1074
SSNACK	Pearson Correlation	-.044	.028	-.021	-.005	.052
	Sig. (2-tailed)	.163	.366	.490	.881	.090
	N	1014	1065	1069	1083	1073
CSNACK	Pearson Correlation	-.033	-.030	-.006	.129**	.087**
	Sig. (2-tailed)	.289	.322	.835	.000	.004
	N	1014	1066	1070	1084	1074
FSNACK	Pearson Correlation	.117**	-.018	-.008	-.031	.042
	Sig. (2-tailed)	.000	.558	.794	.305	.168
	N	1014	1064	1068	1082	1072
TOOTHPAS	Pearson Correlation	1.000	-.063*	-.008	-.071*	-.022
	Sig. (2-tailed)	.	.040	.783	.020	.481
	N	1066	1058	1051	1061	1054
FLUOR	Pearson Correlation	-.063*	1.000	-.162**	.002	-.027
	Sig. (2-tailed)	.040	.	.000	.939	.363
	N	1058	1138	1121	1133	1126
CARE	Pearson Correlation	-.008	-.162**	1.000	-.037	-.012
	Sig. (2-tailed)	.783	.000	.	.213	.684
	N	1051	1121	1141	1137	1128
SHOPTYPE	Pearson Correlation	-.071*	.002	-.037	1.000	.006
	Sig. (2-tailed)	.020	.939	.213	.	.845
	N	1061	1133	1137	1156	1143
BEDTIME	Pearson Correlation	-.022	-.027	-.012	.006	1.000
	Sig. (2-tailed)	.481	.363	.684	.845	.
	N	1054	1126	1128	1143	1148

Correlations

		TOOTHPAS	FLUOR	CARE	SHOPTYPE	BEDTIME
NIGHT	Pearson Correlation	-.066*	.054	-.016	.062*	.058
	Sig. (2-tailed)	.034	.071	.583	.039	.054
	N	1034	1106	1108	1122	1115
WHOBRUSH	Pearson Correlation	.881**	-.091**	.020	-.104**	-.038
	Sig. (2-tailed)	.000	.003	.510	.000	.202
	N	1038	1104	1103	1116	1111

Correlations

		NIGHT	WHOBRUSH
DEPCAT	Pearson Correlation	-.102**	.111**
	Sig. (2-tailed)	.001	.000
	N	1126	1120
SMM	Pearson Correlation	-.023	.020
	Sig. (2-tailed)	.486	.545
	N	902	896
SSM	Pearson Correlation	-.017	.053
	Sig. (2-tailed)	.616	.114
	N	902	896
STM	Pearson Correlation	-.028	.025
	Sig. (2-tailed)	.396	.458
	N	902	896
LACTM	Pearson Correlation	-.039	.030
	Sig. (2-tailed)	.231	.361
	N	925	919
YEASTM	Pearson Correlation	.010	-.014
	Sig. (2-tailed)	.776	.668
	N	890	886
SM	Pearson Correlation	-.048	.002
	Sig. (2-tailed)	.109	.935
	N	1094	1088
SS	Pearson Correlation	.032	-.004
	Sig. (2-tailed)	.293	.906
	N	1094	1088
ST	Pearson Correlation	-.037	.009
	Sig. (2-tailed)	.218	.770
	N	1094	1088
LACT	Pearson Correlation	-.077*	.097**
	Sig. (2-tailed)	.010	.001
	N	1110	1105
YEAST	Pearson Correlation	-.028	-.004
	Sig. (2-tailed)	.360	.899
	N	1083	1077
SEX	Pearson Correlation	.019	.029
	Sig. (2-tailed)	.537	.334
	N	1102	1095
HIGHRISK	Pearson Correlation	.065*	-.199**
	Sig. (2-tailed)	.036	.000
	N	1030	1022
WTCENT	Pearson Correlation	.014	-.039
	Sig. (2-tailed)	.664	.225
	N	951	947
HTCENT	Pearson Correlation	-.010	-.086**
	Sig. (2-tailed)	.762	.008
	N	960	955
WTCENTA	Pearson Correlation	.056	-.122**
	Sig. (2-tailed)	.067	.000
	N	1060	1054
HTCENTA	Pearson Correlation	-.017	-.136**
	Sig. (2-tailed)	.590	.000
	N	1042	1036

Correlations

		NIGHT	WHOBRUSH
WTCENTB	Pearson Correlation	.070*	-.132**
	Sig. (2-tailed)	.022	.000
	N	1060	1054
HTCENTB	Pearson Correlation	.024	-.140**
	Sig. (2-tailed)	.433	.000
	N	1056	1050
CIMMUN	Pearson Correlation	.009	.010
	Sig. (2-tailed)	.772	.750
	N	1068	1061
MEDICAT	Pearson Correlation	.018	-.045
	Sig. (2-tailed)	.548	.144
	N	1075	1068
AGEWEAN	Pearson Correlation	.034	-.035
	Sig. (2-tailed)	.264	.248
	N	1076	1069
BRFEED	Pearson Correlation	.007	.124**
	Sig. (2-tailed)	.828	.000
	N	1087	1081
DUMMY	Pearson Correlation	-.080*	-.103**
	Sig. (2-tailed)	.013	.002
	N	962	955
STILLDMY	Pearson Correlation	.013	-.089*
	Sig. (2-tailed)	.719	.018
	N	728	718
VITAMIN	Pearson Correlation	.009	.066*
	Sig. (2-tailed)	.785	.038
	N	1008	1001
SIBLINGS	Pearson Correlation	-.034	.137**
	Sig. (2-tailed)	.257	.000
	N	1086	1079
MUM_AGE	Pearson Correlation	.047	.006
	Sig. (2-tailed)	.126	.847
	N	1046	1041
MARSTAT	Pearson Correlation	-.055	.120**
	Sig. (2-tailed)	.083	.000
	N	991	979
SC	Pearson Correlation	-.078*	.118**
	Sig. (2-tailed)	.021	.000
	N	879	875
SMOKE	Pearson Correlation	.058	-.117**
	Sig. (2-tailed)	.076	.000
	N	952	945
EMPLOY	Pearson Correlation	-.051	.155**
	Sig. (2-tailed)	.091	.000
	N	1082	1075
BMEAL	Pearson Correlation	-.012	.005
	Sig. (2-tailed)	.692	.865
	N	1125	1119
DMEAL	Pearson Correlation	-.046	.067*
	Sig. (2-tailed)	.121	.025
	N	1125	1119

5.4 Logistic regression analyses

Notes

Output Created		28-FEB-2000 11:48:13
Comments		
Input	Data	H:\DUNDEEC\THESIS\CHAID7.sav
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	697
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing
Syntax		LOGISTIC REGRESSION VAR=d1mft4 /METHOD=FSTEP(COND) depcat highrisk smoke dummy shoptype fsnack mum_age night sex /CRITERIA PIN(.05) POUT(.10) ITERATE(20) CUT(.5) .
Resources	Elapsed Time	0:00:00.44

Case Processing Summary

Unweighted Cases[a]		N	Percent
Selected Cases	Included in Analysis	697	100.0
	Missing Cases	0	.0
	Total	697	100.0
Unselected Cases		0	.0
Total		697	100.0

a. If weight is in effect, see classification table for the total number of cases.

Dependent Variable Encoding

Original Value	Internal Value
0	0
1	1

Block 1: Method = Forward Stepwise (Conditional)

Omnibus Tests of Model Coefficients

		Chi-square	df	Sig.
Step 1	Step	34.052	1	.000
	Block	34.052	1	.000
	Model	34.052	1	.000
Step 2	Step	14.109	1	.000
	Block	48.161	2	.000
	Model	48.161	2	.000
Step 3	Step	9.101	1	.003
	Block	57.262	3	.000
	Model	57.262	3	.000
Step 4	Step	5.841	1	.016
	Block	63.103	4	.000
	Model	63.103	4	.000
Step 5	Step	4.420	1	.036
	Block	67.522	5	.000
	Model	67.522	5	.000

Model Summary

Step	-2 Log likelihood	Cox & Snell R Square	Nagelkerke R Square
1	931.149	.048	.064
2	917.040	.067	.089
3	907.939	.079	.105
4	902.098	.087	.115
5	897.679	.092	.123

Classification Table[a]

Observed			Predicted		
			D1MFT4		Percentage Correct
			0	1	
Step 1	D1MFT4	0	302	60	83.4
		1	215	120	35.8
	Overall Percentage				60.5
Step 2	D1MFT4	0	210	152	58.0
		1	116	219	65.4
	Overall Percentage				61.5
Step 3	D1MFT4	0	285	77	78.7
		1	181	154	46.0
	Overall Percentage				63.0
Step 4	D1MFT4	0	232	130	64.1
		1	127	208	62.1
	Overall Percentage				63.1
Step 5	D1MFT4	0	253	109	69.9
		1	153	182	54.3
	Overall Percentage				62.4

a. The cut value is .500

Variables in the Equation

		B	S.E.	Wald	df	Sig.	Exp(B)
Step 1[a]	HIGHRISK	-1.033	.182	32.363	1	.000	.356
	Constant	1.726	.329	27.590	1	.000	5.617
Step 2[b]	HIGHRISK	-.864	.188	21.193	1	.000	.421
	SMOKE	-.606	.162	14.068	1	.000	.545
	Constant	2.978	.476	39.140	1	.000	19.640
Step 3[c]	HIGHRISK	-.836	.189	19.531	1	.000	.434
	SMOKE	-.596	.163	13.382	1	.000	.551
	NIGHT	-.477	.161	8.774	1	.003	.621
	Constant	4.682	.760	37.904	1	.000	107.974
Step 4[d]	HIGHRISK	-.782	.191	16.807	1	.000	.457
	SMOKE	-.544	.165	10.916	1	.001	.581
	DUMMY	-.431	.179	5.785	1	.016	.650
	NIGHT	-.527	.164	10.346	1	.001	.590
	Constant	5.201	.801	42.154	1	.000	181.473
Step 5[e]	DEPCAT	.350	.166	4.437	1	.035	1.419
	HIGHRISK	-.704	.195	13.076	1	.000	.495
	SMOKE	-.486	.167	8.457	1	.004	.615
	DUMMY	-.406	.180	5.087	1	.024	.666
	NIGHT	-.511	.165	9.622	1	.002	.600
	Constant	2.905	1.343	4.678	1	.031	18.273

a. Variable(s) entered on step 1: HIGHRISK.

b. Variable(s) entered on step 2: SMOKE.

c. Variable(s) entered on step 3: NIGHT.

d. Variable(s) entered on step 4: DUMMY.

e. Variable(s) entered on step 5: DEPCAT.

Model if Term Removed[a]

Variable		Model Log Likelihood	Change in -2 Log Likelihood	df	Sig. of the Change
Step 1	HIGHRISK	-482.615	34.080	1	.000
Step 2	HIGHRISK	-469.460	21.880	1	.000
	SMOKE	-465.579	14.118	1	.000
Step 3	HIGHRISK	-464.019	20.099	1	.000
	SMOKE	-460.680	13.420	1	.000
	NIGHT	-458.525	9.112	1	.003
Step 4	HIGHRISK	-459.664	17.231	1	.000
	SMOKE	-456.510	10.922	1	.001
	DUMMY	-453.972	5.845	1	.016
	NIGHT	-456.449	10.800	1	.001
Step 5	DEPCAT	-451.050	4.421	1	.035
	HIGHRISK	-455.495	13.312	1	.000
	SMOKE	-453.059	8.439	1	.004
	DUMMY	-451.405	5.132	1	.023
	NIGHT	-453.857	10.035	1	.002

a. Based on conditional parameter estimates

Variables not in the Equation

			Score	df	Sig.
Step 1	Variables	DEPCAT	9.241	1	.002
		SMOKE	14.200	1	.000
		DUMMY	6.332	1	.012
		SHOPTYPE	7.674	1	.006
		FSNACK	2.403	1	.121
		MUM_AGE	3.663	1	.056
		NIGHT	9.661	1	.002
		SEX	.277	1	.598
	Overall Statistics		37.887	8	.000
Step 2	Variables	DEPCAT	5.847	1	.016
		DUMMY	4.147	1	.042
		SHOPTYPE	3.209	1	.073
		FSNACK	3.761	1	.052
		MUM_AGE	2.033	1	.154
		NIGHT	8.951	1	.003
		SEX	.204	1	.651
	Overall Statistics		24.141	7	.001
Step 3	Variables	DEPCAT	5.169	1	.023
		DUMMY	5.820	1	.016
		SHOPTYPE	2.888	1	.089
		FSNACK	3.500	1	.061
		MUM_AGE	2.034	1	.154
		SEX	.138	1	.710
	Overall Statistics		15.385	6	.017
Step 4	Variables	DEPCAT	4.452	1	.035
		SHOPTYPE	2.884	1	.089
		FSNACK	3.055	1	.081
		MUM_AGE	1.390	1	.238
		SEX	.055	1	.814
	Overall Statistics		9.654	5	.086
Step 5	Variables	SHOPTYPE	2.040	1	.153
		FSNACK	2.886	1	.089
		MUM_AGE	.450	1	.502
		SEX	.086	1	.769
	Overall Statistics		5.228	4	.265

```
LOGISTIC REGRESSION VAR=d3mft4
  /METHOD=FSTEP(COND) depcat highrisk smoke dummy shoptype fsnack mum_age night sex
  /CRITERIA PIN(.05) POUT(.10) ITERATE(20) CUT(.5) .
```

Logistic Regression

Notes

Output Created		28-FEB-2000 11:48:15
Comments		
Input	Data	H:\DUNDEEC\THESIS\CHAID7.sav
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	697
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing
Syntax		LOGISTIC REGRESSION VAR=d3mft4 /METHOD=FSTEP(COND) depcat highrisk smoke dummy shoptype fsnack mum_age night sex /CRITERIA PIN(.05) POUT(.10) ITERATE(20) CUT(.5) .
Resources	Elapsed Time	0:00:00.39

Case Processing Summary

Unweighted Cases[a]		N	Percent
Selected Cases	Included in Analysis	697	100.0
	Missing Cases	0	.0
	Total	697	100.0
Unselected Cases		0	.0
Total		697	100.0

a. If weight is in effect, see classification table for the total number of cases.

Dependent Variable Encoding

Original Value	Internal Value
0	0
1	1

Block 1: Method = Forward Stepwise (Conditional)

Omnibus Tests of Model Coefficients

		Chi-square	df	Sig.
Step 1	Step	38.146	1	.000
	Block	38.146	1	.000
	Model	38.146	1	.000
Step 2	Step	19.427	1	.000
	Block	57.573	2	.000
	Model	57.573	2	.000
Step 3	Step	10.569	1	.001
	Block	68.141	3	.000
	Model	68.141	3	.000
Step 4	Step	4.617	1	.032
	Block	72.758	4	.000
	Model	72.758	4	.000

Model Summary

Step	-2 Log likelihood	Cox & Snell R Square	Nagelkerke R Square
1	832.607	.053	.075
2	813.180	.079	.111
3	802.612	.093	.131
4	797.995	.099	.139

Classification Table[a]

			Predicted		
			D3MFT4		Percentage Correct
	Observed		0	1	
Step 1	D3MFT4	0	387	89	81.3
		1	130	91	41.2
	Overall Percentage				68.6
Step 2	D3MFT4	0	420	56	88.2
		1	155	66	29.9
	Overall Percentage				69.7
Step 3	D3MFT4	0	415	61	87.2
		1	151	70	31.7
	Overall Percentage				69.6
Step 4	D3MFT4	0	423	53	88.9
		1	164	57	25.8
	Overall Percentage				68.9

a. The cut value is .500

Variables in the Equation

		B	S.E.	Wald	df	Sig.	Exp(B)
Step 1[a]	HIGHRISK	-1.113	.180	38.122	1	.000	.329
	Constant	1.135	.315	12.997	1	.000	3.112
Step 2[b]	HIGHRISK	-.908	.187	23.466	1	.000	.403
	SMOKE	-.767	.174	19.308	1	.000	.465
	Constant	2.708	.484	31.320	1	.000	15.004
Step 3[c]	HIGHRISK	-.878	.189	21.534	1	.000	.416
	SMOKE	-.758	.176	18.555	1	.000	.469
	NIGHT	-.525	.162	10.539	1	.001	.592
	Constant	4.580	.768	35.598	1	.000	97.533
Step 4[d]	DEPCAT	.388	.180	4.625	1	.032	1.474
	HIGHRISK	-.788	.194	16.519	1	.000	.455
	SMOKE	-.693	.179	15.026	1	.000	.500
	NIGHT	-.510	.162	9.847	1	.002	.601
	Constant	2.068	1.389	2.217	1	.136	7.906

a. Variable(s) entered on step 1: HIGHRISK.

b. Variable(s) entered on step 2: SMOKE.

c. Variable(s) entered on step 3: NIGHT.

d. Variable(s) entered on step 4: DEPCAT.

Model if Term Removed[a]

Variable		Model Log Likelihood	Change in -2 Log Likelihood	df	Sig. of the Change
Step 1	HIGHRISK	-435.437	38.267	1	.000
Step 2	HIGHRISK	-418.255	23.329	1	.000
	SMOKE	-416.357	19.534	1	.000
Step 3	HIGHRISK	-412.004	21.397	1	.000
	SMOKE	-410.690	18.769	1	.000
	NIGHT	-406.600	10.589	1	.001
Step 4	DEPCAT	-401.310	4.624	1	.032
	HIGHRISK	-407.188	16.381	1	.000
	SMOKE	-406.563	15.130	1	.000
	NIGHT	-403.951	9.907	1	.002

a. Based on conditional parameter estimates

Variables not in the Equation

			Score	df	Sig.
Step 1	Variables	DEPCAT	9.345	1	.002
		SMOKE	19.691	1	.000
		DUMMY	1.945	1	.163
		SHOPTYPE	6.258	1	.012
		FSNACK	.320	1	.571
		MUM_AGE	.854	1	.355
		NIGHT	11.695	1	.001
		SEX	2.859	1	.091
	Overall Statistics		40.474	8	.000
Step 2	Variables	DEPCAT	5.331	1	.021
		DUMMY	.671	1	.413
		SHOPTYPE	1.706	1	.191
		FSNACK	1.011	1	.315
		MUM_AGE	.113	1	.736
		NIGHT	10.853	1	.001
		SEX	2.821	1	.093
	Overall Statistics		21.273	7	.003
Step 3	Variables	DEPCAT	4.642	1	.031
		DUMMY	1.431	1	.232
		SHOPTYPE	1.425	1	.233
		FSNACK	.910	1	.340
		MUM_AGE	.091	1	.763
		SEX	2.656	1	.103
	Overall Statistics		10.604	6	.101
Step 4	Variables	DUMMY	1.086	1	.297
		SHOPTYPE	.877	1	.349
		FSNACK	.806	1	.369
		MUM_AGE	.058	1	.810
		SEX	2.856	1	.091
Overall Statistics			6.015	5	.305

HIGH RISK

dlmft >= 3.

? dlmft - 3+

```
recode depcat(1,2,3,4,5=5)/housing(2,3,4=2)/
 smoke (1,2=2)/shoptype(1,2,4=2).
LOGISTIC REGRESSION VAR=target1   -
  /METHOD=FSTEP(COND) highrisk fvessel smoke housing snack shoptype vitamin depc
   at cvessel fsnack
  /CRITERIA PIN(.05) POUT(.10) ITERATE(20) CUT(.5) .

Logistic Regression

Notes
```

Output Created		08 Oct 99 11:23:37
Comments		
Input	Data	H:\DUNDEEC\THESIS\CHAID4.sav
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	784
Syntax		LOGISTIC REGRESSION VAR=target1 /METHOD=FSTEP(COND) highrisk fvessel smoke housing snack shoptype vitamin depcat cvessel fsnack /CRITERIA PIN(.05) POUT(.10) ITERATE(20) CUT(.5) .
Resources	Elapsed Time	0:00:00.86

¢

□

```
      Total number of cases:      784 (Unweighted)
      Number of selected cases:   784
      Number of unselected cases: 0

      Number of selected cases:                    784
      Number rejected because of missing data:     0
      Number of cases included in the analysis:    784


Dependent Variable Encoding:

Original        Internal
Value           Value
1               0
2               1
```

□

```
Dependent Variable..   TARGET1    dlmft target

Beginning Block Number  0.  Initial Log Likelihood Function

-2 Log Likelihood    970.68582

* Constant is included in the model.

Estimation terminated at iteration number 3 because
parameter estimates changed by less than .001

Classification Table for TARGET1
The Cut Value is .50
                      Predicted
                    0-2      3+      Percent Correct
                      0  ó    3
Observed          ôôôôôôôôôôôôôôôôô
```

0-2	0	541	0	100.00%		
3+	3	243	0	.00%		
			Overall	69.01%		

---------- Variables in the Equation ----------

Variable	B	S.E.	Wald	df	Sig	R	Exp(B)
Constant	-.8004	.0772	107.4128	1	.0000		

Beginning Block Number 1. Method: Forward Stepwise (COND)

---------- Variables not in the Equation ----------

Residual Chi Square 81.123 with 10 df Sig = .0000

Variable	Score	df	Sig	R
HIGHRISK	38.4673	1	.0000	.1938
FVESSEL	21.7150	1	.0000	.1425
SMOKE	26.0204	1	.0000	.1573
HOUSING	45.0273	1	.0000	.2105
SNACK	4.9255	1	.0265	.0549
SHOPTYPE	26.8035	1	.0000	.1599
VITAMIN	4.2305	1	.0397	.0479
DEPCAT	18.3498	1	.0000	.1298
CVESSEL	.9702	1	.3246	.0000
FSNACK	.9892	1	.3199	.0000

Variable(s) Entered on Step Number
1.. HOUSING

Estimation terminated at iteration number 3 because
Log Likelihood decreased by less than .01 percent.

-2 Log Likelihood	925.549
Goodness of Fit	783.996
Cox & Snell - R^2	.056
Nagelkerke - R^2	.079

	Chi-Square	df	Significance
Model	45.137	1	.0000
Block	45.137	1	.0000
Step	45.137	1	.0000

Classification Table for TARGET1
The Cut Value is .50

		Predicted 0-2	Predicted 3+	Percent Correct
Observed				
0-2	0	541	0	100.00%
3+	3	243	0	.00%
			Overall	69.01%

---------- Variables in the Equation ----------

Variable	B	S.E.	Wald	df	Sig	R	Exp(B)
HOUSING	1.0545	.1597	43.5787	1	.0000	.2070	2.8706
Constant	-2.3784	.2593	84.1123	1	.0000		

---------- Model if Term Removed ----------

Based on Conditional Parameter Estimates

Term Removed	Log Likelihood	-2 Log LR	df	Significance of Log LR
HOUSING	-485.566	45.583	1	.0000

3.. FVESSEL

Estimation terminated at iteration number 3 because
Log Likelihood decreased by less than .01 percent.

-2 Log Likelihood	901.997
Goodness of Fit	779.143
Cox & Snell - R^2	.084
Nagelkerke - R^2	.118

	Chi-Square	df	Significance
Model	68.688	3	.0000
Block	68.688	3	.0000
Step	9.223	1	.0024

Classification Table for TARGET1
The Cut Value is .50

		Predicted 0-2	Predicted 3+	Percent Correct
Observed				
0-2	0	515	26	95.19%
3+	3	203	40	16.46%
			Overall	70.79%

---------- Variables in the Equation ----------

Variable	B	S.E.	Wald	df	Sig	R	Exp(B)
HIGHRISK	-.6123	.1882	10.5798	1	.0011	-.0940	.5421
FVESSEL	.5484	.1791	9.3708	1	.0022	.0871	1.7304
HOUSING	.7833	.1735	20.3860	1	.0000	.1376	2.1888
Constant	-1.6266	.5531	8.6493	1	.0033		

---------- Model if Term Removed ----------
Based on Conditional Parameter Estimates

Term Removed	Log Likelihood	-2 Log LR	df	Significance of Log LR
HIGHRISK	-456.235	10.473	1	.0012
FVESSEL	-455.617	9.236	1	.0024
HOUSING	-461.277	20.557	1	.0000

---------- Variables not in the Equation ----------
Residual Chi Square 12.742 with 7 df Sig = .0786

Variable	Score	df	Sig	R
SMOKE	3.9130	1	.0479	.0444
SNACK	3.5357	1	.0601	.0398
SHOPTYPE	2.7931	1	.0947	.0286
VITAMIN	.2057	1	.6502	.0000
DEPCAT	1.0100	1	.3149	.0000
CVESSEL	.4312	1	.5114	.0000
FSNACK	2.8401	1	.0919	.0294

Variable(s) Entered on Step Number
4.. SMOKE

Estimation terminated at iteration number 3 because
Log Likelihood decreased by less than .01 percent.

-2 Log Likelihood	898.129
Goodness of Fit	778.214
Cox & Snell - R^2	.088
Nagelkerke - R^2	.124

	Chi-Square	df	Significance
Model	72.557	4	.0000

---------- Variables not in the Equation ----------
Residual Chi Square 36.384 with 9 df Sig = .0000

Variable	Score	df	Sig	R
HIGHRISK	14.6558	1	.0001	.1142
FVESSEL	13.4518	1	.0002	.1086
SMOKE	7.7724	1	.0053	.0771
SNACK	3.3772	1	.0661	.0377
SHOPTYPE	4.5507	1	.0329	.0513
VITAMIN	.8615	1	.3533	.0000
DEPCAT	2.1363	1	.1438	.0118
CVESSEL	.3895	1	.5326	.0000
FSNACK	1.2630	1	.2611	.0000

Variable(s) Entered on Step Number
2.. HIGHRISK

Estimation terminated at iteration number 3 because
Log Likelihood decreased by less than .01 percent.

-2 Log Likelihood	911.220
Goodness of Fit	781.108
Cox & Snell - R^2	.073
Nagelkerke - R^2	.103

	Chi-Square	df	Significance
Model	59.465	2	.0000
Block	59.465	2	.0000
Step	14.328	1	.0002

Classification Table for TARGET1
The Cut Value is .50

		Predicted 0-2	Predicted 3+	Percent Correct
Observed	0-2	0	464	85.77%
	3+	3	164	32.51%
			Overall	69.26%

---------- Variables in the Equation ----------

Variable	B	S.E.	Wald	df	Sig	R	Exp(B)
HIGHRISK	-.7038	.1850	14.4674	1	.0001	-.1133	.4947
HOUSING	.8185	.1723	22.5607	1	.0000	.1455	2.2671
Constant	-.8191	.4822	2.8856	1	.0894		

---------- Model if Term Removed ----------
Based on Conditional Parameter Estimates

Term Removed	Log Likelihood	-2 Log LR	df	Significance of Log LR
HIGHRISK	-462.782	14.343	1	.0002
HOUSING	-467.015	22.809	1	.0000

---------- Variables not in the Equation ----------
Residual Chi Square 22.025 with 8 df Sig = .0049

Variable	Score	df	Sig	R
FVESSEL	9.4600	1	.0021	.0877
SMOKE	4.8207	1	.0281	.0539
SNACK	2.9140	1	.0878	.0307
SHOPTYPE	2.6861	1	.1012	.0266
VITAMIN	.2078	1	.6485	.0000
DEPCAT	1.1383	1	.2860	.0000
CVESSEL	.1859	1	.6664	.0000
FSNACK	2.3263	1	.1272	.0183

Variable(s) Entered on Step Number

Block	72.557	4	.0000	
Step	3.869	1	.0492	

Classification Table for TARGET1
The Cut Value is .50

		Predicted 0-2	3+	Percent Correct
Observed		0	3	
0-2	0	515	26	95.19%
3+	3	203	40	16.46%
			Overall	70.79%

SENS = 16%
SPEC = 95%

---------- Variables in the Equation ----------

Variable	B	S.E.	Wald	df	Sig	R	Exp(B)
HIGHRISK	-.5554	.1909	8.4678	1	.0036	-.0816	.5738
FVESSEL	.5234	.1800	8.4572	1	.0036	.0816	1.6878
SMOKE	-.3472	.1760	3.8929	1	.0485	-.0442	.7067
HOUSING	.6767	.1818	13.8486	1	.0002	.1105	1.9674
Constant	-.6579	.7391	.7923	1	.3734		

---------- Model if Term Removed ----------
Based on Conditional Parameter Estimates

Term Removed	Log Likelihood	-2 Log LR	df	Significance of Log LR
HIGHRISK	-453.254	8.378	1	.0038
FVESSEL	-453.233	8.337	1	.0039
SMOKE	-451.001	3.873	1	.0491
HOUSING	-456.002	13.875	1	.0002

□

---------- Variables not in the Equation ----------
Residual Chi Square 8.899 with 6 df Sig = .1793

Variable	Score	df	Sig	R
SNACK	3.1142	1	.0776	.0339
SHOPTYPE	2.0085	1	.1564	.0030
VITAMIN	.1046	1	.7464	.0000
DEPCAT	.9397	1	.3324	.0000
CVESSEL	.1456	1	.7028	.0000
FSNACK	3.5863	1	.0583	.0404

No more variables can be deleted or added.

High risk

$d_3 mft \geq 3$

```
LOGISTIC REGRESSION VAR=target3
  /METHOD=FSTEP(COND) highrisk fvessel smoke housing snack shoptype vitamin depc
   at cvessel fsnack
  /CRITERIA PIN(.05) POUT(.10) ITERATE(20) CUT(.5) .
```

Logistic Regression

Notes

Output Created		08 Oct 99 11:25:48
Comments		
Input	Data	H:\DUNDEEC\THESIS\CHAID4.sav
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	784
Syntax		LOGISTIC REGRESSION VAR=target3 /METHOD=FSTEP(COND) highrisk fvessel smoke housing snack shoptype vitamin depcat cvessel fsnack /CRITERIA PIN(.05) POUT(.10) ITERATE(20) CUT(.5) .
Resources	Elapsed Time	0:00:00.52

```
      Total number of cases:      784 (Unweighted)
      Number of selected cases:   784
      Number of unselected cases: 0

      Number of selected cases:                   784
      Number rejected because of missing data:    0
      Number of cases included in the analysis:   784
```

Dependent Variable Encoding:

Original Value	Internal Value
1	0
2	1

Dependent Variable.. TARGET3 d3mft target

Beginning Block Number 0. Initial Log Likelihood Function

-2 Log Likelihood 759.60594

* Constant is included in the model.

Estimation terminated at iteration number 3 because
Log Likelihood decreased by less than .01 percent.

Classification Table for TARGET3
The Cut Value is .50

		Predicted 0-2	Predicted 3+	Percent Correct
Observed				
0-2	0	636	0	100.00%
3+	3	148	0	.00%

```
                ôòòòòòòòòòòòòòòò
                          Overall  81.12%

---------------------- Variables in the Equation -----------------------

Variable            B        S.E.     Wald    df      Sig         R    Exp(B)

Constant      -1.4579      .0913 255.2077     1     .0000

Beginning Block Number  1.  Method: Forward Stepwise (COND)

□

--------------- Variables not in the Equation -----------------
Residual Chi Square     74.659 with     10 df     Sig =  .0000

    Variable            Score    df      Sig         R

    HIGHRISK          37.5126     1     .0000    .2162
    FVESSEL           17.1751     1     .0000    .1413
    SMOKE             19.6381     1     .0000    .1524
    HOUSING           36.0868     1     .0000    .2118
    SNACK              5.4387     1     .0197    .0673
    SHOPTYPE          25.5039     1     .0000    .1759
    VITAMIN             .1351     1     .7132    .0000
    DEPCAT            17.2778     1     .0000    .1418
    CVESSEL             .0000     1     .9963    .0000
    FSNACK              .1630     1     .6865    .0000

    Variable(s) Entered on Step Number
    1..        HIGHRISK

    Estimation terminated at iteration number 4 because
    parameter estimates changed by less than .001

     -2 Log Likelihood          725.295
     Goodness of Fit            784.000
     Cox & Snell - R^2             .043
     Nagelkerke - R^2              .069

                        Chi-Square     df Significance

     Model                  34.311      1          .0000
     Block                  34.311      1          .0000
     Step                   34.311      1          .0000

    Classification Table for TARGET3
    The Cut Value is .50
                          Predicted
                          0-2      3+      Percent Correct
                            0  ó    3
    Observed              ôòòòòòòòòòòòòòòò
       0-2          0     ó  636  ó    0  ó  100.00%
                          ôòòòòòòòòòòòòòòò
       3+           3     ó  148  ó    0  ó     .00%
                          ôòòòòòòòòòòòòòòò
                                Overall  81.12%

---------------------- Variables in the Equation -----------------------

Variable            B        S.E.     Wald    df      Sig         R    Exp(B)

HIGHRISK      -1.1405      .1918  35.3665     1     .0000   -.2096     .3197
Constant        .4548      .3227   1.9870     1     .1587

□

----------------- Model if Term Removed -----------------
        Based on Conditional Parameter Estimates

Term          Log                                    Significance
Removed       Likelihood     -2 Log LR     df        of Log LR

HIGHRISK       -380.158         35.022      1             .0000

--------------- Variables not in the Equation -----------------
Residual Chi Square     38.623 with      9 df     Sig =  .0000
```

Variable	Score	df	Sig	R
FVESSEL	8.2381	1	.0041	.0906
SMOKE	8.2086	1	.0042	.0904
HOUSING	16.7446	1	.0000	.1393
SNACK	4.3292	1	.0375	.0554
SHOPTYPE	11.1364	1	.0008	.1097
VITAMIN	.5878	1	.4433	.0000
DEPCAT	7.4369	1	.0064	.0846
CVESSEL	.1699	1	.6802	.0000
FSNACK	1.0342	1	.3092	.0000

Variable(s) Entered on Step Number
2.. HOUSING

Estimation terminated at iteration number 4 because
Log Likelihood decreased by less than .01 percent.

-2 Log Likelihood	708.764
Goodness of Fit	776.259
Cox & Snell - R^2	.063
Nagelkerke - R^2	.101

	Chi-Square	df	Significance
Model	50.842	2	.0000
Block	50.842	2	.0000
Step	16.531	1	.0000

Classification Table for TARGET3
The Cut Value is .50

		Predicted 0-2	Predicted 3+	Percent Correct
Observed				
0-2	0	636	0	100.00%
3+	3	148	0	.00%
			Overall	81.12%

□

---------- Variables in the Equation ----------

Variable	B	S.E.	Wald	df	Sig	R	Exp(B)
HIGHRISK	-.8012	.2081	14.8247	1	.0001	-.1299	.4488
HOUSING	.8404	.2086	16.2372	1	.0001	.1369	2.3173
Constant	-1.4030	.5628	6.2153	1	.0127		

---------- Model if Term Removed ----------
Based on Conditional Parameter Estimates

Term Removed	Log Likelihood	-2 Log LR	df	Significance of Log LR
HIGHRISK	-361.743	14.722	1	.0001
HOUSING	-362.786	16.807	1	.0000

---------- Variables not in the Equation ----------
Residual Chi Square 22.127 with 8 df Sig = .0047

Variable	Score	df	Sig	R
FVESSEL	6.6027	1	.0102	.0778
SMOKE	2.8730	1	.0901	.0339
SNACK	3.5451	1	.0597	.0451
SHOPTYPE	3.0511	1	.0807	.0372
VITAMIN	1.6603	1	.1976	.0000
DEPCAT	1.6597	1	.1976	.0000
CVESSEL	.3027	1	.5822	.0000
FSNACK	.8061	1	.3693	.0000

Variable(s) Entered on Step Number
3.. FVESSEL

Estimation terminated at iteration number 4 because

Log Likelihood decreased by less than .01 percent.

-2 Log Likelihood	702.392
Goodness of Fit	779.548
Cox & Snell - R^2	.070
Nagelkerke - R^2	.113

	Chi-Square	df	Significance
Model	57.214	3	.0000
Block	57.214	3	.0000
Step	6.373	1	.0116

Classification Table for TARGET3
The Cut Value is .50

		Predicted 0-2	Predicted 3+	Percent Correct
Observed				
0-2	0	636	0	100.00%
3+	3	148	0	.00%
			Overall	81.12%

---------- Variables in the Equation ----------

Variable	B	S.E.	Wald	df	Sig	R	Exp(B)
HIGHRISK	-.7131	.2114	11.3812	1	.0007	-.1111	.4901
FVESSEL	.5203	.2035	6.5402	1	.0105	.0773	1.6826
HOUSING	.8047	.2094	14.7654	1	.0001	.1296	2.2360
Constant	-2.1749	.6416	11.4893	1	.0007		

---------- Model if Term Removed ----------
Based on Conditional Parameter Estimates

Term Removed	Log Likelihood	-2 Log LR	df	Significance of Log LR
HIGHRISK	-356.834	11.276	1	.0008
FVESSEL	-354.393	6.395	1	.0114
HOUSING	-358.810	15.228	1	.0001

---------- Variables not in the Equation ----------
Residual Chi Square 15.428 with 7 df Sig = .0309

Variable	Score	df	Sig	R
SMOKE	2.2958	1	.1297	.0197
SNACK	3.9788	1	.0461	.0510
SHOPTYPE	3.1493	1	.0760	.0389
VITAMIN	1.6699	1	.1963	.0000
DEPCAT	1.5325	1	.2157	.0000
CVESSEL	2.0107	1	.1562	.0038
FSNACK	1.0168	1	.3133	.0000

Variable(s) Entered on Step Number
4.. SNACK

Estimation terminated at iteration number 4 because
Log Likelihood decreased by less than .01 percent.

-2 Log Likelihood	697.947
Goodness of Fit	780.747
Cox & Snell - R^2	.076
Nagelkerke - R^2	.122

	Chi-Square	df	Significance
Model	61.659	4	.0000
Block	61.659	4	.0000
Step	4.444	1	.0350

Classification Table for TARGET3
The Cut Value is .50

Observed		Predicted 0-2	Predicted 3+	Percent Correct
0-2	0	636	0	100.00%
3+	3	148	0	.00%
			Overall	81.12%

SENS - 0%
SPEC = 100%.

---------- Variables in the Equation ----------

Variable	B	S.E.	Wald	df	Sig	R	Exp(B)
HIGHRISK	-.6913	.2129	10.5405	1	.0012	-.1060	.5009
FVESSEL	.5444	.2050	7.0514	1	.0079	.0815	1.7235
HOUSING	.7829	.2105	13.8336	1	.0002	.1248	2.1877
SNACK	-.7297	.3725	3.8367	1	.0501	-.0492	.4821
Constant	-1.4121	.7516	3.5295	1	.0603		

---------- Model if Term Removed ----------
Based on Conditional Parameter Estimates

Term Removed	Log Likelihood	-2 Log LR	df	Significance of Log LR
HIGHRISK	-354.192	10.438	1	.0012
FVESSEL	-352.422	6.897	1	.0086
HOUSING	-356.089	14.230	1	.0002
SNACK	-351.208	4.469	1	.0345

---------- Variables not in the Equation ----------
Residual Chi Square 11.598 with 6 df Sig = .0716

Variable	Score	df	Sig	R
SMOKE	1.9736	1	.1601	.0000
SHOPTYPE	2.8096	1	.0937	.0326
VITAMIN	2.0608	1	.1511	.0089
DEPCAT	1.3931	1	.2379	.0000
CVESSEL	2.3754	1	.1233	.0222
FSNACK	.2360	1	.6271	.0000

No more variables can be deleted or added.

5.5 CHAID analyses

D1MFT4

Cat.	%	n
0	52.82	150
1	47.18	134
Total	(100.00)	284

DEPCAT

P-value=0.0005; Chi-square=22.7334; df=3

1

Cat.	%	n
0	81.08	30
1	18.92	7
Total	(13.03)	37

2,3;4

Cat.	%	n
0	57.00	57
1	43.00	43
Total	(35.21)	100

NIGHT

P-value=0.0022; Chi-square=9.3617; df=1

drink

Cat.	%	n
0	23.53	4
1	**76.47**	**13**
Total	(5.99)	17

neither

Cat.	%	n
0	63.86	53
1	36.14	30
Total	(29.23)	83

DUMMY

P-value=0.0021; Chi-square=9.5007; df=1

yes

Cat.	%	n
0	50.98	26
1	49.02	25
Total	(17.96)	51

no

Cat.	%	n
0	84.38	27
1	15.63	5
Total	(11.27)	32

5

Cat.	%	n
0	15.38	2
1	**84.62**	**11**
Total	(4.58)	13

6

Cat.	%	n
0	45.52	61
1	**54.48**	**73**
Total	(47.18)	134

HOUSING

P-value=0.0011; Chi-square=14.3412; df=1

own;social rent

Cat.	%	n
0	40.65	50
1	**59.35**	**73**
Total	(43.31)	123

private rent;other

Cat.	%	n
0	100.00	11
1	0.00	0
Total	(3.87)	11

SMEAL

P-value=0.0010; Chi-square=10.8969; df=1

yes

Cat.	%	n
0	25.42	15
1	**74.58**	**44**
Total	(20.77)	59

no

Cat.	%	n
0	54.69	35
1	45.31	29
Total	(22.54)	64

D1MFT4

Cat.	%	n
0	53.95	273
1	46.05	233
Total	(100.00)	506

BRFEED

P-value=0.0000; Chi-square=27.5918; df=1

yes

Cat.	%	n
0	67.12	149
1	32.88	73
Total	(43.87)	222

no

Cat.	%	n
0	43.66	124
1	**56.34**	**160**
Total	(56.13)	284

HIGHRISK

P-value=0.0006; Chi-square=11.6895; df=1

yes

Cat.	%	n
0	28.89	26
1	**71.11**	**64**
Total	(17.79)	90

SMOKE

P-value=0.0097; Chi-square=9.2693; df=2

both

Cat.	%	n
0	43.75	14
1	**56.25**	**18**
Total	(6.32)	32

one

Cat.	%	n
0	9.68	3
1	**90.32**	**28**
Total	(6.13)	31

none

Cat.	%	n
0	33.33	9
1	**66.67**	**18**
Total	(5.34)	27

no

Cat.	%	n
0	50.52	98
1	49.48	96
Total	(38.34)	194

DUMMY

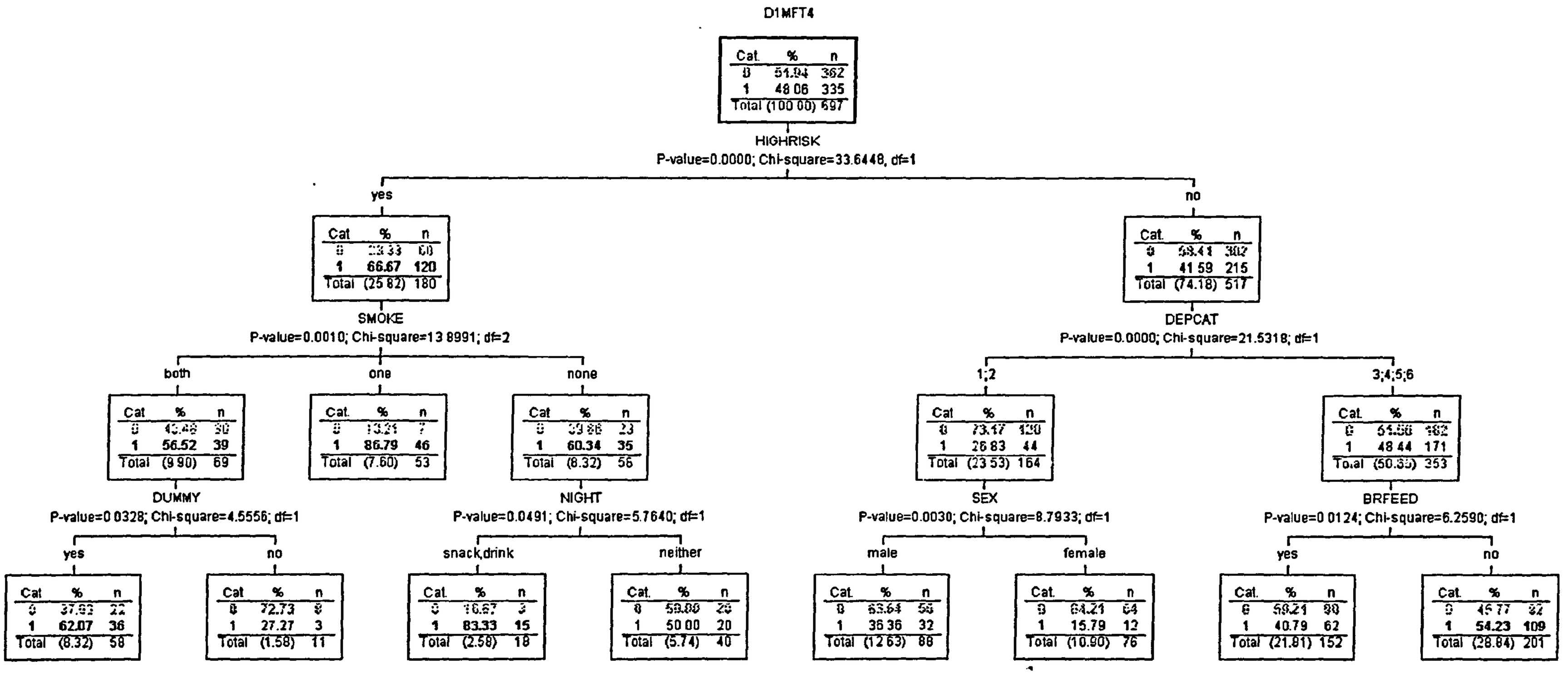
P-value=0.0110; Chi-square=6.4612; df=1

yes

Cat.	%	n
0	45.14	65
1	**54.86**	**79**
Total	(28.46)	144

no

Cat.	%	n
0	66.00	33
1	34.00	17
Total	(9.88)	50


D1MFT4
Cat. % n
0 51.94 362
1 48.06 335
Total (100.00) 697
HIGHRISK
P-value=0.0000; Chi-square=33.6448, df=1
yes
Cat % n
0 33.33 60
1 66.67 120
Total (25.82) 180
SMOKE
P-value=0.0010; Chi-square=13.8991; df=2
both
Cat % n
0 43.48 30
1 56.52 39
Total (9.90) 69
DUMMY
P-value=0.0328; Chi-square=4.5556; df=1
yes
Cat % n
0 37.93 22
1 62.07 36
Total (8.32) 58
no
Cat % n
0 72.73 8
1 27.27 3
Total (1.58) 11
one
Cat % n
0 13.21 7
1 86.79 46
Total (7.60) 53
none
Cat % n
0 39.66 23
1 60.34 35
Total (8.32) 58
NIGHT
P-value=0.0491; Chi-square=5.7640; df=1
snack,drink
Cat % n
0 16.67 3
1 83.33 15
Total (2.58) 18
neither
Cat % n
0 50.00 20
1 50.00 20
Total (5.74) 40
no
Cat % n
0 58.41 302
1 41.59 215
Total (74.18) 517
DEPCAT
P-value=0.0000; Chi-square=21.5318; df=1
1;2
Cat % n
0 73.17 120
1 26.83 44
Total (23.53) 164
SEX
P-value=0.0030; Chi-square=8.7933; df=1
male
Cat % n
0 63.64 56
1 36.36 32
Total (12.63) 88
female
Cat % n
0 84.21 64
1 15.79 12
Total (10.90) 76
3;4;5;6
Cat % n
0 51.58 182
1 48.44 171
Total (50.65) 353
BRFEED
P-value=0.0124; Chi-square=6.2590; df=1
yes
Cat % n
0 59.21 90
1 40.79 62
Total (21.91) 152
no
Cat % n
0 45.77 92
1 54.23 109
Total (28.84) 201

D3MFT4

Cat.	%	n
0	67.96	193
1	32.04	91
Total	(100.00)	284

SHOPTYPE

P-value=0.0017; Chi-square=13.4624; df=1

foot;bus;taxi

Cat.	%	n
0	50.68	37
1	49.32	36
Total	(25.70)	73

DUMMY

P-value=0.0107; Chi-square=6.5158; df=1

yes

Cat.	%	n
0	41.51	22
1	**58.49**	**31**
Total	(18.66)	53

BVESSEL

P-value=0.0237; Chi-square=5.1134; df=1

yes

Cat.	%	n
0	27.59	8
1	**72.41**	**21**
Total	(10.21)	29

no

Cat.	%	n
0	58.33	14
1	41.67	10
Total	(8.45)	24

no

Cat.	%	n
0	75.00	15
1	25.00	5
Total	(7.04)	20

car

Cat.	%	n
0	73.93	156
1	26.07	55
Total	(74.30)	211

LACTM

P-value=0.0127; Chi-square=6.2059; df=1

negative

Cat.	%	n
0	79.70	106
1	20.30	27
Total	(46.83)	133

NIGHT

P-value=0.0148; Chi-square=7.9008; df=1

snack;drink

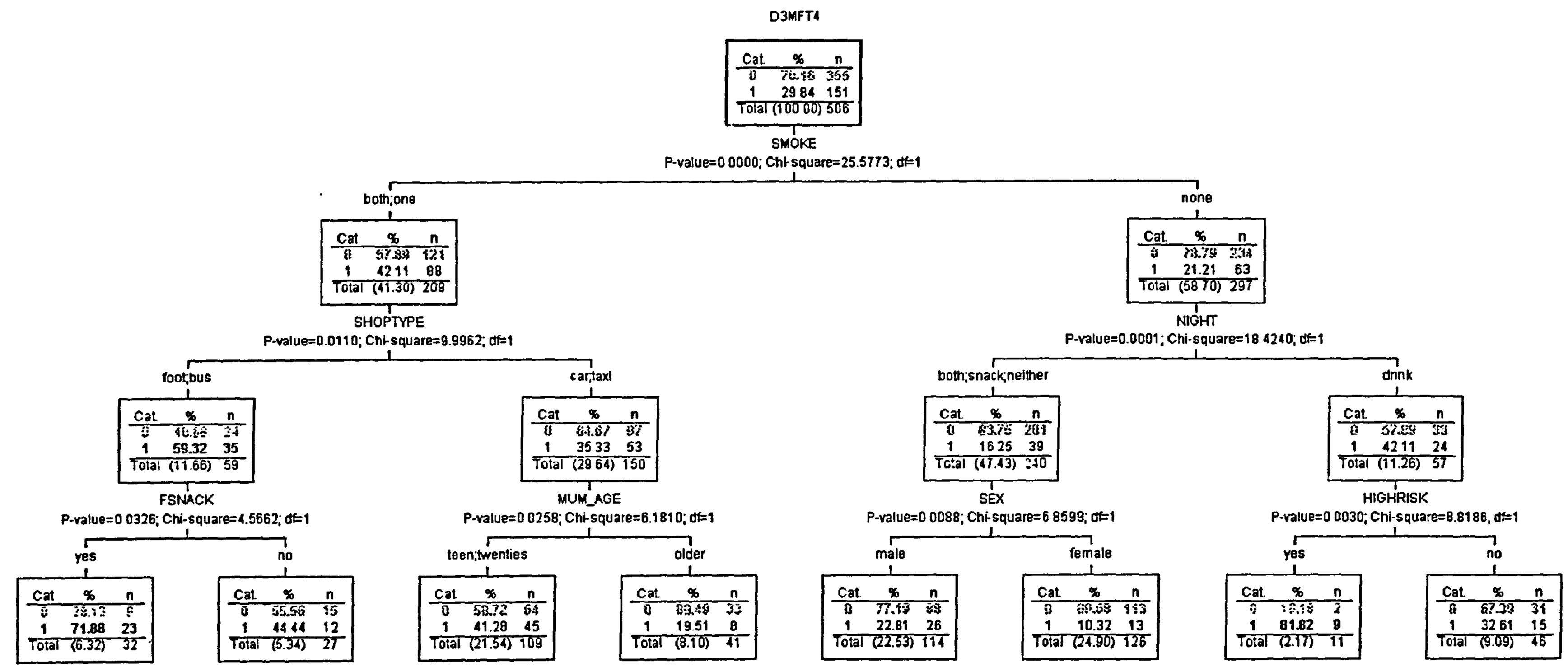
Cat.	%	n
0	60.71	17
1	39.29	11
Total	(9.86)	28

neither

Cat.	%	n
0	84.76	89
1	15.24	16
Total	(36.97)	105

positive

Cat.	%	n
0	64.10	50
1	35.90	28
Total	(27.46)	78


D3MFT4
Cat. % n
0 70.16 355
1 29.84 151
Total (100.00) 506
SMOKE
P-value=0.0000; Chi-square=25.5773; df=1
both;one
Cat. % n
0 57.89 121
1 42.11 88
Total (41.30) 209
SHOPTYPE
P-value=0.0110; Chi-square=9.9962; df=1
foot;bus
Cat. % n
0 40.68 24
1 59.32 35
Total (11.66) 59
FSNACK
P-value=0.0326; Chi-square=4.5662; df=1
yes
Cat. % n
0 28.13 9
1 71.88 23
Total (6.32) 32
no
Cat. % n
0 55.56 15
1 44.44 12
Total (5.34) 27
car;taxi
Cat. % n
0 64.67 97
1 35.33 53
Total (29.64) 150
MUM_AGE
P-value=0.0258; Chi-square=6.1810; df=1
teen;twenties
Cat. % n
0 58.72 64
1 41.28 45
Total (21.54) 109
older
Cat. % n
0 80.49 33
1 19.51 8
Total (8.10) 41
none
Cat. % n
0 78.79 234
1 21.21 63
Total (58.70) 297
NIGHT
P-value=0.0001; Chi-square=18.4240; df=1
both;snack;neither
Cat. % n
0 83.75 201
1 16.25 39
Total (47.43) 240
SEX
P-value=0.0088; Chi-square=6.8599; df=1
male
Cat. % n
0 77.19 88
1 22.81 26
Total (22.53) 114
female
Cat. % n
0 89.68 113
1 10.32 13
Total (24.90) 126
drink
Cat. % n
0 57.89 33
1 42.11 24
Total (11.26) 57
HIGHRISK
P-value=0.0030; Chi-square=8.8186, df=1
yes
Cat. % n
0 18.18 2
1 81.82 9
Total (2.17) 11
no
Cat. % n
0 67.39 31
1 32.61 15
Total (9.09) 46

D3MFT4

Cat.	%	n
0	68.29	476
1	31.71	221
Total	(100.00)	697

HIGHRISK

P-value=0.0000; Chi-square=39.8128; df=1

yes

Cat.	%	n
0	49.44	89
1	50.56	91
Total	(25.82)	180

no

Cat.	%	n
0	74.85	387
1	25.15	130
Total	(74.18)	517

SMOKE

P-value=0.0000; Chi-square=19.4037; df=1

both;one

Cat.	%	n
0	63.87	122
1	36.13	69
Total	(27.40)	191

NIGHT

P-value=0.0048; Chi-square=9.9526; df=1

snack;drink

Cat.	%	n
0	44.68	21
1	55.32	26
Total	(6.74)	47

neither

Cat.	%	n
0	70.14	101
1	29.86	43
Total	(20.66)	144

none

Cat.	%	n
0	81.29	265
1	18.71	61
Total	(46.77)	326

DEPCAT

P-value=0.0424; Chi-square=6.9291; df=1

1;2;3;4

Cat.	%	n
0	85.71	174
1	14.29	29
Total	(29.12)	203

5;6

Cat.	%	n
0	73.98	91
1	26.02	32
Total	(17.65)	123

d1mfttarget

Cat	%	n
0-2	71.83	204
3+	28 17	80
Total	(100 00)	284

SHOPTYPE
P value=0.0001; Chi-square=18 9916, df=1

foot,bus,taxi

Cat	%	n
0-2	52.05	38
3+	47 95	35
Total	(25 70)	73

FSNACK
P value=0.0403; Chi-square=4 2039, df=1

yes

Cat	%	n
0-2	41 46	17
3+	58 54	24
Total	(14 44)	41

DUMMY
P-value=0 0352, Chi-square=4 4375, df=1

yes

Cat	%	n
0-2	32 26	10
3+	67.74	21
Total	(10.92)	31

SMEAL
P-value=0 0072, Chi-square=7.2346, df=1

yes

Cat	%	n
0-2	11 76	2
3+	88 24	15
Total	(5 99)	17

no

Cat	%	n
0 2	57.14	8
3+	42 86	6
Total	(4 93)	14

no

Cat	%	n
0-2	70.00	7
3+	30 00	3
Total	(3 52)	10

no

Cat	%	n
0-2	65.63	21
3+	34 38	11
Total	(11.27)	32

car

Cat	%	n
0-2	79 87	166
3+	21 33	45
Total	(74 30)	211

HIGHRISK
P-value=0.0020; Chi-square=9 5178, df=1

yes

Cat	%	n
0-2	58.82	20
3+	41 18	14
Total	(11 97)	34

NIGHT
P-value=0 0076, Chi-square=7.1238, df=1

drink

Cat	%	n
0 2	80.00	8
3+	20 00	2
Total	(3 52)	10

neither

Cat	%	n
0 2	100.00	34
3+	0 00	0
Total	(11 97)	34

no

Cat	%	n
0-2	82.49	146
3+	17 51	31
Total	(62.32)	177

LACTM
P-value=0 0171, Chi square=5 6813, df=1

negative

Cat	%	n
0-2	87.61	99
3+	12 39	14
Total	(39 79)	113

VITAMIN
P-value=0 0433, Chi-square=4 0845, df=1

yes

Cat	%	n
0 2	95.45	42
3+	4 55	2
Total	(15 49)	44

SEX
P-value=0 0499, Chi-square=3 8460, df=1

male

Cat	%	n
0 2	73.53	25
3+	26 47	9
Total	(11.97)	34

CSNACK
P-value=0 0447, Chi-square=4 0296, df=1

yes

Cat	%	n
0-2	50.00	5
3+	50 00	5
Total	(3.52)	10

FSNACK
P-value=0 0095, Chi-square=6 7200, df=1

yes

Cat	%	n
0 2	100.00	14
3+	0 00	0
Total	(4 93)	14

no

Cat	%	n
0-2	60.00	6
3+	40 00	4
Total	(3 52)	10

no

Cat	%	n
0 2	83.33	20
3+	16 67	4
Total	(8 45)	24

female

Cat	%	n
0 2	91.43	32
3+	8 57	3
Total	(12 32)	35

positive

Cat	%	n
0 2	73.44	47
3+	26 56	17
Total	(22.54)	64

SMEAL
P-value=0.0183, Chi-square=5 5652, df=1

yes

Cat	%	n
0 2	57.69	15
3+	42 31	11
Total	(9 15)	26

no

Cat	%	n
0-2	84.21	32
3+	15 79	6
Total	(13 38)	38

DUMMY
P-value=0.0017, Chi-square=9 7969, df=1

yes

Cat	%	n
0 2	100.00	22
3+	0 00	0
Total	(7.75)	22

no

Cat	%	n
0 2	62.50	10
3+	37 50	6
Total	(5 63)	16

d1mfi target

Cat.	%	n
0-2	72.33	366
3+	27.67	140
Total	(100.00)	506

HIGHRISK
P-value=0.0000; Chi-square=28.5373; df=1

yes

Cat.	%	n
0-2	52.63	60
3+	47.37	54
Total	(22.53)	114

FVESSEL
P-value=0.0283; Chi-square=4.8121; df=1

yes

Cat.	%	n
0-2	61.54	40
3+	38.46	25
Total	(12.85)	65

no

Cat.	%	n
0-2	40.82	20
3+	59.18	29
Total	(9.68)	49

SMOKE
P-value=0.0043; Chi-square=10.6840; df=1

both

Cat.	%	n
0-2	65.22	15
3+	34.78	8
Total	(4.55)	23

one;none

Cat.	%	n
0-2	19.23	5
3+	80.77	21
Total	(5.14)	26

no

Cat.	%	n
0-2	78.06	306
3+	21.94	86
Total	(77.47)	392

HOUSING
P-value=0.0004; Chi-square=16.6985; df=1

own;private rent;other

Cat.	%	n
0-2	82.62	252
3+	17.38	53
Total	(60.28)	305

FVESSEL
P-value=0.0060; Chi-square=7.5650; df=1

yes

Cat.	%	n
0-2	85.48	212
3+	14.52	36
Total	(49.01)	248

no

Cat.	%	n
0-2	70.18	40
3+	29.82	17
Total	(11.26)	57

social rent

Cat.	%	n
0-2	62.07	54
3+	37.93	33
Total	(17.19)	87

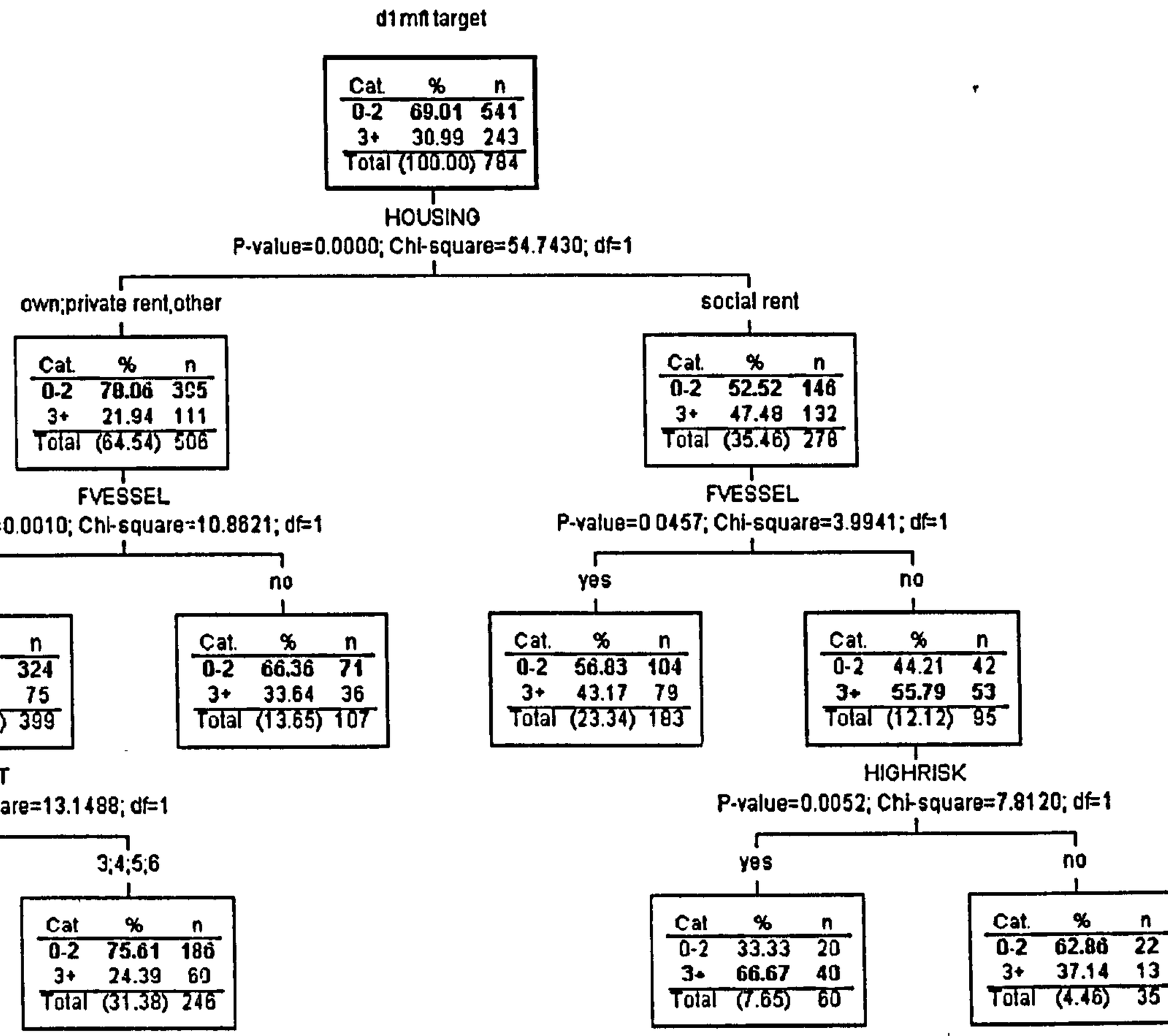
SNACK
P-value=0.0086; Chi-square=6.9048; df=1

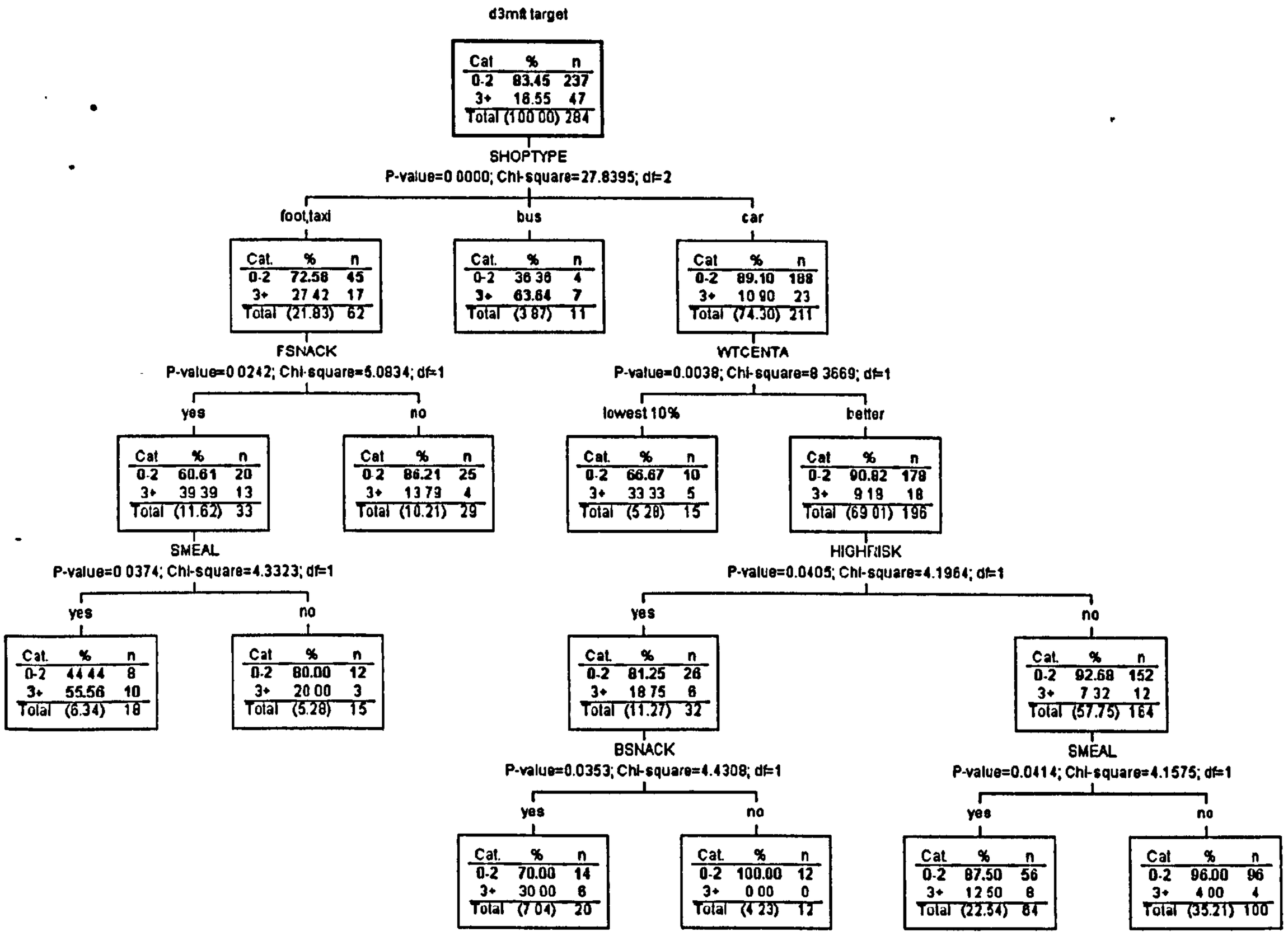
yes

Cat.	%	n
0-2	57.14	44
3+	42.86	33
Total	(15.22)	77

no

Cat.	%	n
0-2	100.00	10
3+	0.00	0
Total	(1.98)	10


d1mft target
Cat. % n
0-2 69.01 541
3+ 30.99 243
Total (100.00) 784
HOUSING
P-value=0.0000; Chi-square=54.7430; df=1
own;private rent,other
Cat. % n
0-2 78.06 395
3+ 21.94 111
Total (64.54) 506
FVESSEL
P-value=0.0010; Chi-square=10.8621; df=1
yes
Cat. % n
0-2 81.20 324
3+ 18.80 75
Total (50.69) 399
DEPCAT
P-value=0.0014; Chi-square=13.1488; df=1
1;2
Cat % n
0-2 90.20 138
3+ 9.80 15
Total (19.52) 153
3;4;5;6
Cat % n
0-2 75.61 186
3+ 24.39 60
Total (31.38) 246
no
Cat. % n
0-2 66.36 71
3+ 33.64 36
Total (13.65) 107
social rent
Cat. % n
0-2 52.52 146
3+ 47.48 132
Total (35.46) 278
FVESSEL
P-value=0.0457; Chi-square=3.9941; df=1
yes
Cat. % n
0-2 56.83 104
3+ 43.17 79
Total (23.34) 183
no
Cat. % n
0-2 44.21 42
3+ 55.79 53
Total (12.12) 95
HIGHRISK
P-value=0.0052; Chi-square=7.8120; df=1
yes
Cat % n
0-2 33.33 20
3+ 66.67 40
Total (7.65) 60
no
Cat. % n
0-2 62.86 22
3+ 37.14 13
Total (4.46) 35


d3mft target
Cat % n
0-2 83.45 237
3+ 16.55 47
Total (100.00) 284
SHOPTYPE
P-value=0.0000; Chi-square=27.8395; df=2
foot,taxi
bus
car
Cat. % n
0-2 72.58 45
3+ 27.42 17
Total (21.83) 62
Cat. % n
0-2 36.36 4
3+ 63.64 7
Total (3.87) 11
Cat % n
0-2 89.10 188
3+ 10.90 23
Total (74.30) 211
FSNACK
P-value=0.0242; Chi-square=5.0834; df=1
WTCENTA
P-value=0.0038; Chi-square=8.3669; df=1
yes
no
lowest 10%
better
Cat % n
0-2 60.61 20
3+ 39.39 13
Total (11.62) 33
Cat % n
0-2 86.21 25
3+ 13.79 4
Total (10.21) 29
Cat. % n
0-2 66.67 10
3+ 33.33 5
Total (5.28) 15
Cat % n
0-2 90.82 178
3+ 9.18 18
Total (69.01) 196
SMEAL
P-value=0.0374; Chi-square=4.3323; df=1
HIGHRISK
P-value=0.0405; Chi-square=4.1964; df=1
yes
no
Cat. % n
0-2 44.44 8
3+ 55.56 10
Total (6.34) 18
Cat % n
0-2 80.00 12
3+ 20.00 3
Total (5.28) 15
Cat. % n
0-2 81.25 26
3+ 18.75 6
Total (11.27) 32
Cat. % n
0-2 92.68 152
3+ 7.32 12
Total (57.75) 164
BSNACK
P-value=0.0353; Chi-square=4.4308; df=1
SMEAL
P-value=0.0414; Chi-square=4.1575; df=1
yes
no
Cat. % n
0-2 70.00 14
3+ 30.00 6
Total (7.04) 20
Cat. % n
0-2 100.00 12
3+ 0.00 0
Total (4.23) 12
Cat % n
0-2 87.50 56
3+ 12.50 8
Total (22.54) 64
Cat % n
0-2 96.00 96
3+ 4.00 4
Total (35.21) 100

d3mft target

Cat.	%	n
0-2	**83.40**	**422**
3+	16.60	84
Total	(100.00)	506

SHOPTYPE

P-value=0.0002; Chi-square=21.9197; df=2

foot;taxi

Cat.	%	n
0-2	**74.26**	**75**
3+	25.74	26
Total	(19.96)	101

CVESSEL

P-value=0.0386; Chi-square=4.2794; df=1

yes

Cat.	%	n
0-2	**100.00**	**11**
3+	0.00	0
Total	(2.17)	11

no

Cat.	%	n
0-2	**71.11**	**64**
3+	28.89	26
Total	(17.79)	90

FSNACK

P-value=0.0233; Chi-square=5.1469; df=1

yes

Cat.	%	n
0-2	**59.52**	**25**
3+	40.48	17
Total	(8.30)	42

no

Cat.	%	n
0-2	**81.25**	**39**
3+	18.75	9
Total	(9.49)	48

bus

Cat.	%	n
0-2	**55.00**	**11**
3+	45.00	9
Total	(3.95)	20

car

Cat.	%	n
0-2	**87.27**	**336**
3+	12.73	49
Total	(76.09)	385

HIGHRISK

P-value=0.0001; Chi-square=14.9615; df=1

yes

Cat.	%	n
0-2	**72.13**	**44**
3+	27.87	17
Total	(12.06)	61

no

Cat.	%	n
0-2	**90.12**	**292**
3+	9.88	32
Total	(64.03)	324

DEPCAT

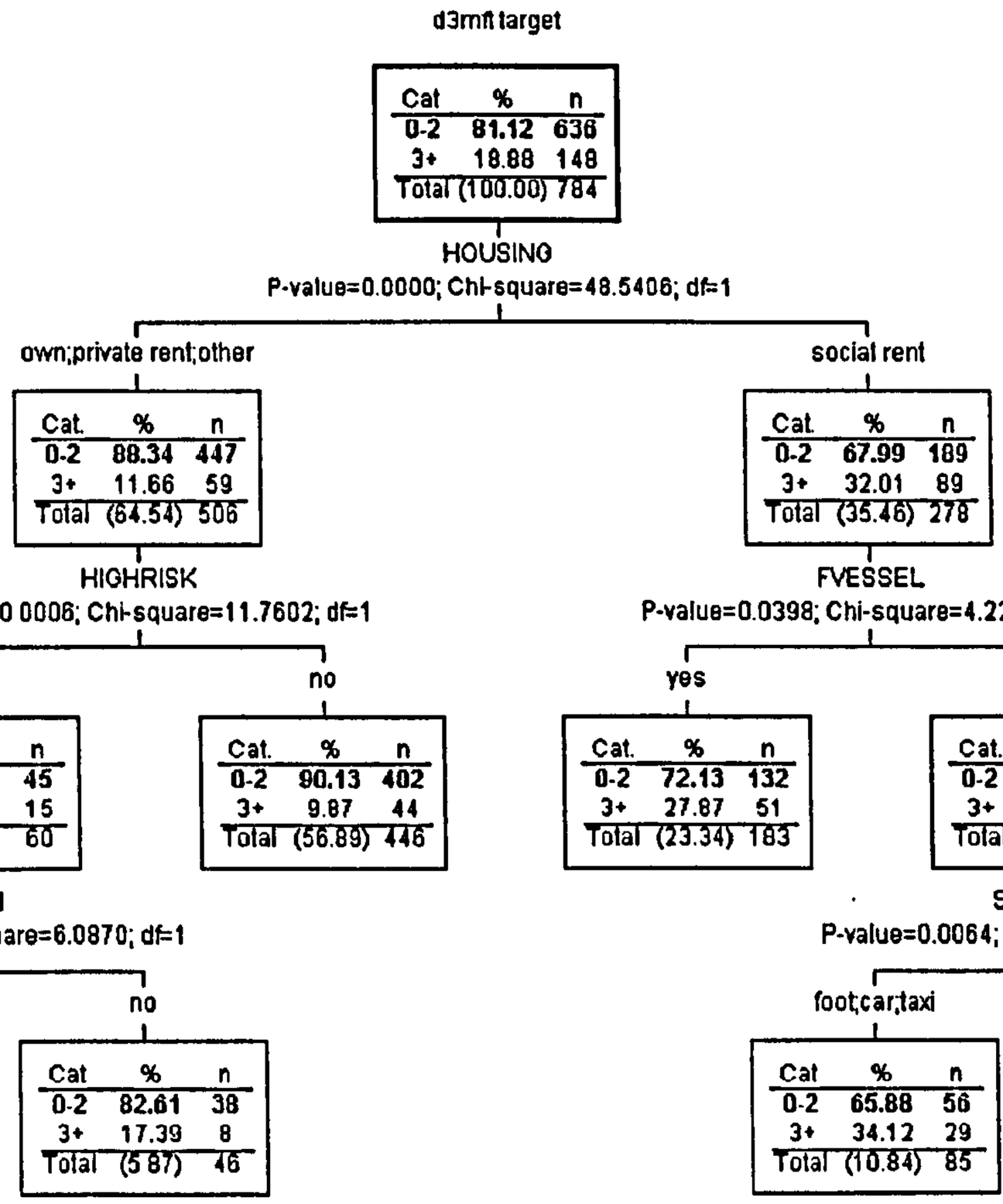
P-value=0.0282; Chi-square=9.6605; df=1

1;2;3;4;5

Cat.	%	n
0-2	**93.87**	**199**
3+	6.13	13
Total	(41.90)	212

6

Cat.	%	n
0-2	**83.04**	**93**
3+	16.96	19
Total	(22.13)	112


d3mft target
Cat % n
0-2 81.12 636
3+ 18.88 148
Total (100.00) 784
HOUSING
P-value=0.0000; Chi-square=48.5406; df=1
own;private rent;other
Cat. % n
0-2 88.34 447
3+ 11.66 59
Total (64.54) 506
social rent
Cat. % n
0-2 67.99 189
3+ 32.01 89
Total (35.46) 278
HIGHRISK
P-value=0 0006; Chi-square=11.7602; df=1
yes
Cat. % n
0-2 75.00 45
3+ 25.00 15
Total (7.65) 60
no
Cat. % n
0-2 90.13 402
3+ 9.87 44
Total (56.89) 446
FVESSEL
P-value=0.0398; Chi-square=4.2284; df=1
yes
Cat. % n
0-2 72.13 132
3+ 27.87 51
Total (23.34) 183
no
Cat. % n
0-2 60.00 57
3+ 40.00 38
Total (12.12) 95
VITAMIN
P-value=0.0136; Chi-square=6.0870; df=1
yes
Cat % n
0-2 50.00 7
3+ 50.00 7
Total (1.79) 14
no
Cat % n
0-2 82.61 38
3+ 17.39 8
Total (5 87) 46
SHOPTYPE
P-value=0.0064; Chi-square=11.6422; df=1
foot;car;taxi
Cat % n
0-2 65.88 56
3+ 34.12 29
Total (10.84) 85
bus
Cat. % n
0-2 10.00 1
3+ 90.00 9
Total (1.28) 10

5.6 Analyses for representativeness of data

Was subject consented?

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	yes	1703	90.3	90.3	90.3
	no	182	9.7	9.7	100.0
	Total	1885	100.0	100.0	

Year 4 clinical exam

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	yes	1365	72.4	80.2	80.2
	no	338	17.9	19.8	100.0
	Total	1703	90.3	100.0	
Missing	System	182	9.7		
Total		1885	100.0		

In 784 model

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	yes	784	41.6	57.4	57.4
	no	581	30.8	42.6	100.0
	Total	1365	72.4	100.0	
Missing	System	520	27.6		
Total		1885	100.0		

Crosstab

			DEPCAT grouping						Total
			1	2	3	4	5	6+7	
Was subject consented?	yes	Count	192	184	118	231	84	893	1702
		% within DEPCAT grouping	93.7%	91.1%	92.2%	90.9%	87.5%	89.4%	90.3%
	no	Count	13	18	10	23	12	106	182
		% within DEPCAT grouping	6.3%	8.9%	7.8%	9.1%	12.5%	10.6%	9.7%
Total		Count	205	202	128	254	96	999	1884
		% within DEPCAT grouping	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	5.246[a]	5	.387
Likelihood Ratio	5.509	5	.357
Linear-by-Linear Association	4.042	1	.044
N of Valid Cases	1884		

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 9.27.

Crosstab

			DEPCAT grouping						Total
			1	2	3	4	5	6+7	
Year 4 clinical exam	yes	Count	150	152	102	197	65	698	1364
		% within DEPCAT grouping	78.1%	82.6%	86.4%	85.3%	77.4%	78.2%	80.1%
	no	Count	42	32	16	34	19	195	338
		% within DEPCAT grouping	21.9%	17.4%	13.6%	14.7%	22.6%	21.8%	19.9%
Total		Count	192	184	118	231	84	893	1702
		% within DEPCAT grouping	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	10.568[a]	5	.061
Likelihood Ratio	11.089	5	.050
Linear-by-Linear Association	1.941	1	.164
N of Valid Cases	1702		

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 16.68.

Crosstab

			DEPCAT grouping						Total
			1	2	3	4	5	6	
In 784 model	yes	Count	97	101	64	115	35	372	784
		% within DEPCAT grouping	64.7%	66.4%	62.7%	58.4%	53.8%	53.3%	57.5%
	no	Count	53	51	38	82	30	326	580
		% within DEPCAT grouping	35.3%	33.6%	37.3%	41.6%	46.2%	46.7%	42.5%
Total		Count	150	152	102	197	65	698	1364
		% within DEPCAT grouping	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	14.745[a]	5	.012
Likelihood Ratio	14.905	5	.011
Linear-by-Linear Association	13.906	1	.000
N of Valid Cases	1364		

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 27.64.

Report

In 784 model		D1MFT4	D3MFT4
yes	Mean	2.2691	1.3827
	N	784	784
no	Mean	2.4682	1.4337
	N	581	581
Total	Mean	2.3538	1.4044
	N	1365	1365

Ranks

	In 784 model	N	Mean Rank	Sum of Ranks
D1MFT4	yes	784	671.58	526517.50
	no	581	698.41	405777.50
	Total	1365		
D3MFT4	yes	784	677.96	531518.50
	no	581	689.80	400776.50
	Total	1365		

Test Statistics[a]

	D1MFT4	D3MFT4
Mann-Whitney U	218797.50	223798.50
Wilcoxon W	526517.50	531518.50
Z	-1.334	-.658
Asymp. Sig. (2-tailed)	.182	.511

a. Grouping Variable: In 784 model

5.7 Summary of numbers of valid and missing data sets from study questionnaires, microbiological saliva sampling and dental examination used for risk model development.

Summary of numbers of valid and missing data sets from study questionnaires, microbiological saliva sampling and dental examination which were used for risk model development

Statistics

		DEPCAT	SMM	SSM	STM	LACTM	YEASTM	SM
N	Valid	1364	951	951	951	975	938	1168
	Missing	1	414	414	414	390	427	197

Statistics

		SS	ST	LACT	YEAST	SEX	HIGHRISK	WTCENT
N	Valid	1168	1168	1186	1157	1180	1171	1021
	Missing	197	197	179	208	185	194	344

Statistics

		HTCENT	WTCENTA	HTCENTA	WTCENTB	HTCENTB	CIMMUN	MEDICAT
N	Valid	1027	1134	1114	1132	1127	1145	1152
	Missing	338	231	251	233	238	220	213

Statistics

		AGEWEAN	BRFEED	DUMMY	STILLDMY	VITAMIN	SIBLINGS	MUM_AGE
N	Valid	1147	1164	1027	780	1079	1164	1122
	Missing	218	201	338	585	286	201	243

Statistics

		MARSTAT	HOUSING	SC	SMOKE	EMPLOY	BMEAL	DMEAL
N	Valid	1060	1168	931	1014	1159	1159	1159
	Missing	305	197	434	351	206	206	206

Statistics

		TMEAL	SMEAL	NMEAL	BVESSEL	FVESSEL	CVESSEL	SNACK
N	Valid	1158	1156	1157	1147	1145	1145	1117
	Missing	207	209	208	218	220	220	248

6.0 Published abstracts and publications arising from the work of this thesis

Publications and Abstracts

Ballantyne HM, Longbottom C, Pitts NB, Radford JR and Robertson M. (1995): Use of health visitors to access Scottish 1 year olds for caries-risk assessment. Journal of Dental Research 74: 1337 (Abstract)

Radford JR, Ballantyne, HM, Longbottom, C, Pitts, NB, Robertson, M and Pitts, NB. (1995): Prevalence of caries-associated microorganisms in 1-year-old infants from Dundee. Journal of Dental Research, 74: 872 (Abstract 407)

Ballantyne HM, Longbottom C, Pitts NB, Radford JR, Robertson M and Nugent Z. (1996): Factors associated with high caries-risk in Scottish 1 year olds. Journal of Dental Research 75: 1140 (Abstract 81).

Radford, JR, Ballantyne, HM, Longbottom, C, Pitts, NB, Robertson, M and Nugent, ZJ. (1996): Caries associated microorganisms in 1-year olds with caries. Journal of Dental Research 75 (5): 1134 (Abstract 37).

Ballantyne, HM, Longbottom C, Pitts NB, Radford JR and Robertson M (1997): Accessing infants for caries-risk assessment, differences at 1 and 2 years. J. Dent Res 1997; 76: 1034 (Abstract 123)

Longbottom, C, Ballantyne, HM, Pitts, NB, Radford, JR and Robertson, M. (1997): Social factors associated with caries-risk in Scottish 2-year olds. Journal of Dental Research 76: 1034 (Abstract 124).

Longbottom, C, Ballantyne, HM, Pitts, NB, Radford, JR and Robertson, M. (1997): Identification of high caries risk markers in Scottish 1 and 2 year olds using parental questionnaires. Caries Research 31: 307 (Abstract 81)

Radford, JR, Ballantyne, HM, Beighton, D, Longbottom, C, Pitts, NB and Robertson, M. (1997): Isolation frequencies of caries associated microorganisms in infants and their mothers. Journal of Dental Research 76: 1027 (Abstract 71).

Ballantyne HM, Nugent ZJ, Robertson M, Longbottom, C, Radford JR, Beighton D and Pitts, NB. (1998): Risk markers for future caries in infants. Caries Research 32: 106 (Abstract)

Radford, JR, Nugent, ZJ, Ballantyne, HM, Longbottom, C, Pitts, NB, Robertson, M and Beighton, D. (1998): Microbial markers for caries in infants. Journal of Dental Research 77: 654 (Abstract 181).

MacRitchie, HM, Longbottom, NB, Nugent, ZJ, Radford, JR and Robertson, M. (2000): Distribution of caries monitored annually from ages 1 to 4 years in a Scottish cohort. Caries Research – in press.

Radford, JR, Ballantyne, HM, Nugent, ZJ, Beighton, D, Robertson, M and Pitts, NB. (2000): Caries associated microorganisms in infants from different socio-economic backgrounds in Scotland. Journal of Dentistry **28**, 307-312.

H M BALLANTYNE *, C LONGBOTTOM, N B PITTS, J R RADFORD and
M ROBERTSON (Department of Dental Health, University of Dundee, UK):

Use of Health Visitors to access Scottish 1 year olds for caries-risk assessment.

This project was part of a study to identify high caries-risk Scottish infants using microbiological, dental and social factors. The aim was to investigate the practicability of using Health Visitors as a method of gaining access to 1 year old infants for microbiological sampling and dental examination. Fifty eight Health Visitors carried out saliva sampling on 1 year olds using the tongue-loop method (Beighton D, Br. Dent J 160: 329, 1986). Clinical dental examinations (including transillumination) were completed by the research dentist. The Health Visitors and dentist employed various means of access: joint home visits (JHV); joint clinic visits (JCV); separate home visits (SHV) or separate clinic visits (SCV). The method of access was chosen by the Health Visitor, was often area dependent and was tailored to suit both parent and child. Consent to participate in the study was obtained for 701 (83%) of the 842 infants born in Dundee between 1 April and 31 August 1993. Health Visitors sampled 630 infants (90% of those consented), of whom 618 (88% of those consented) were also examined dentally. The access methods employed for these 630 children were: JHV - 165 (26%); JCV - 122 (19%); SHV - 284 (45%); SCV - 9 (1 %) , while 35 (5%) were accessed using other means. Thus Health Visitors, integrating these activities with the demands of their existing caseload, gained access to 90% of the consented cohort.

In conclusion, using various access methods, Health Visitors and a research dentist were able to sample and examine 88% of consented 1 year old infants born between 1 April and 31 August 1993 in Dundee.

Supported by Chief Scientist Office, The Scottish Office Home and Health Department.

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J R RADFORD*, H M BALLANTYNE, C LONGBOTTOM, N B PITTS, M ROBERTSON and D BEIGHTON (Dept of Dental Health, Univ of Dundee, UK and Kings Coll Sch of Med & Dent, UK):

Prevalence of caries-associated micro-organisms in 1 year old infants from Dundee.

Few studies have examined the prevalence of mutans streptococci and lactobacilli in infants. The aim of this study therefore was to determine the prevalence of these caries-associated micro-organisms in the saliva of 1 year old infants. Such information will form the baseline data for a longitudinal study examining the role of the microbiological factors, amongst others, as markers of future caries-risk. Using Health Visitors, a tongue-loop sample was collected from 76 one year old infants resident throughout Dundee. Samples were placed in 1 ml of transport medium dispersed by vortexing and plated on BMSA (for mutans streptococci) and Rogosa agar (for lactobacilli). After incubation, colonies were counted, then characterised using the Gram stain and catalase test. Representatives were stored for confirmatory and later detailed taxonomic identification. The lowest detection level for each of the bacterial groups was 10^3 colony forming units/ml of saliva. Mutans streptococci were recovered from 17% (when detected, range 1.00×10^3 - 8.20×10^4, median 2.00×10^3) and lactobacilli from 9% (range 1.00×10^3 - 2.40×10^4, median 3.00×10^3) of the infants. Therefore, even at 1 year of age these infants are harbouring cariogenic micro-organisms. Subsequent monitoring will determine if these markers can be used as predictors of caries activity.

It is concluded that mutans streptococci and lactobacilli were isolated from the saliva of 17% and 9% respectively, of 1 year old infants. The presence of these micro-organisms may indicate that these children are at risk of developing dental caries.

Supported by the Chief Scientist Office, Scottish Office Home and Health Department.

Factors associated with high caries-risk in Scottish 1 year olds. H M BALLANTYNE*, C LONGBOTTOM, N B PITTS, J R RADFORD, M ROBERTSON and Z NUGENT. (Dept. of Dental Health, University of Dundee, UK)

The aim of this project was to explore a range of factors to identify markers which can predict those infants at highest risk of developing dental caries. It is part of an ongoing longitudinal study of pre-school children. Access to the infants at 1 year of age was gained through the 58 health visitors in Dundee who completed a social and medical questionnaire and facilitated the completion of a parental questionnaire providing data on the infants' oral and dietary habits Microbiological sampling was also carried out and is reported elsewhere. Clinical dental examination at the caries into enamel (D1) threshold, including transillumination with a pen-light, was carried out by a research dentist. Of the 1974 infants born between 1 April 1993 and 31 March 1994, 1747 were consented to participate in the project (88.5%). Questionnaires and dental examination were completed for 1372 of the consented infants (78.5%). Of these 1372 infants, 36 had caries (D1). The significant factors (chi-square test) positively associated with caries in these infants were: bedtime snacking ($P<0.05$); parental smoking ($P<0.01$); housing ($P<0.05$) and health visitors assessment ($P<0.01$). Use of a dummy ($P<0.05$) and married parents ($P<0.001$) were negatively associated with caries. Non-significant factors included toothbrushing, number of snacks and mother's age (t-test). In conclusion, a number of specific social and oral factors have been identified which, together with a health visitor's assessment, can be used to predict those 1 year old Scottish infants who are at high risk of developing dental caries.

Supported by Chief Scientist Office, Scottish Home and Health Department

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Caries-associated micro-organisms in 1 year olds with caries. JR RADFORD*, HM BALLANTYNE, C LONGBOTTOM, NB PITTS, M ROBERTSON, Z NUGENT AND D BEIGHTON[1] (Univ. of Dundee and KCSMD, London[1], UK)

The aim of this study was to compare the frequency of isolation of selected caries-associated micro-organisms recovered from the saliva of 1 year old infants with dental caries (D1-threshold:caries into enamel) with those who were clinically caries-free. 1372 consented infants, living in Dundee, had salivary samples taken (tongue-loop) for microbiological culture and were examined dentally. From this cohort, 36 infants were diagnosed with dental caries (D1) and a matched random sample of 36 caries-free infants was collected for comparative purposes. *Streptococcus mutans* was isolated significantly more frequently from those infants with caries compared with those who were caries-free (31% v. 3%, P=0.004 - Chi square test). In contrast, there were no significant differences (Fisher's Exact test:two tail) between the isolation frequencies of *Streptococcus sobrinus*, lactobacilli and yeasts (1,11 and 14% respectively) in the two groups of infants. This supports the observation that *S. mutans*, in contrast to the other selected caries-associated micro-organisms, is associated with the initiation of caries. All 1372 infants are being studied over four years which should establish whether *S. mutans* will indeed provide a marker for future caries activity.
It is concluded that *S. mutans* was isolated significantly more frequently from the saliva of 1 year old infants with caries (D1) compared to a matched sample that was caries-free.

Supported by the Scottish Office, Home and Health Department. Grant no. K/OPR/2/2/D150

Accessing infants for caries-risk assessment, differences at 1 and 2 years.

HM BALLANTYNE*, C LONGBOTTOM, NB PITTS, JR RADFORD and M ROBERTSON. (Dept. of Dental Health, University of Dundee, UK)

This study was part of a longitudinal project in partnership with health visitors to access and identify high caries-risk infants. The aim was to explore the relative success of different methods of accessing infants at age 2 compared to age 1. All 54 health visitors for Dundee participated and having obtained consent from the parent or guardian, accessed the infants for microbiological saliva sampling and questionnaire completion (caries-risk assessment). Dental examination was carried out by the study dentist. The health visitors and study dentist used several methods to access the infants including a joint visit to the home or clinic or a separate visit for dental examination to the home or clinic. The number of visits required to achieve access was recorded. 88% (1592) of the infants born in one calendar year in Dundee consented to participate. 1428 were seen as one year olds and 1414 as two year olds. Half of the infants at 1 year were accessed jointly, half separately. At 2 years, 28% jointly and 66% separately. This difference was statistically significant ($p<0.001$). The majority of infants (97%) were accessed within 4 visits. 42 and 178 infants exhibited decay at 1 and 2 years respectively. For those infants with decay the most frequent access method was a separate home visit. These results may influence targeting of preventive strategies to infants in the future particularly in terms of the ability to access high caries children successfully. In conclusion, health visitors and a study dentist, using various methods, accessed 90% (1428) and 89% (1414) of consented infants at ages 1 and 2 respectively and the proportion of separate visits increased between the two years.

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124 Social factors associated with caries-risk in Scottish 2 year-olds.

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This study was part of a longitudinal project in partnership with health visitors to identify markers of caries-risk in infants. The aims were, with the infant cohort at 2 years of age to: (1) assess the association between certain social factors and caries experience and (2) assess the sensitivity and specificity of the health visitors' subjective assessment of caries-risk. 1592 of the infants born in Dundee in one calendar year consented to participate. 1414 were seen at 2 years of age. Social data was collected using a questionnaire administered by health visitors, who were also asked to asses if each infant was or was not at high caries-risk. Dental examinations were carried out by a study dentist, with caries recorded at the D1 (caries in enamel) threshold. 178 infants (13% of the cohort) exhibited caries. Social factors significantly associated with caries were: increasing number of siblings; mother a smoker; father a smoker; social class of father; and nature of housing (Chi square - $P<0.01$). The sensitivity of the health visitors' subjective assessment of caries-risk was 64% and the specificity was 76%.

It is concluded that a number of social factors are significantly associated with caries in 2 year-old Scottish infants and that health visitors' subjective judgement may be useful for caries-risk assessment.

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Identification of High Caries-Risk Markers in Scottish 1 and 2 Year Olds Using Parental Questionnaires

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This project was a part of a longitudinal study to identify high caries-risk Scottish infants using microbiological, dental and social factors. The aim of this project was to identify, from parental questionnaires, markers which can predict those infants at highest risk of developing dental caries. Of the 1974 infants born in Dundee between April 1st 1993 and March 31st 1994, 1,592 consented to participate in the study. Dental examinations were carried out by a study dentist (at the D1 – caries in enamel – threshold) at 1 and 2 years of age. The questionnaires sought information on: feeding habits, toothbrushing, fluoride use, child-care attendance and methods of access to food shopping. 1,428 and 1,414 infants were dentally examined at 1 and 2 years, respectively. 1,418 and 1,358 parental questionnaires were completed at 1 and 2 years, respectively. 40 and 173 infants (3 and 13% of the cohort) exhibited caries at 1 and 2 years, respectively. The significant factors (in the 1-year questionnaire data) positively associated with caries (at age 2 years) were: child ate supper daily ($p<0.05$); use of a bottle ($p<0.001$); crisp snack ($p<0.001$); drink during night ($p<0.001$), and toothbrushing by child ($p<0.05$). There was a significant association between caries and the method of transport for food shopping ($p<0.001$) – 'on foot' and 'by bus' being more frequently associated with caries than 'by car' or 'by taxi'. Significant factors negatively correlated with caries were: breastfeeding ($p<0.001$); use of a feeding cup ($p<0.001$); toothbrushing ($p<0.001$), and toothbrushing by parent ($p<0.05$). It is concluded that for Scottish infants a number of markers of high caries-risk can be identified from parental questionnaires administered when the child is 1 year old.

71 Isolation frequencies of caries-associated micro-organisms in infants and their mothers. JR RADFORD*, HM BALLANTYNE, D BEIGHTON[1], C LONGBOTTOM, NB PITTS, M ROBERTSON & ZJ NUGENT (Univ. of Dundee and [1]KCSMD London, UK)

The aim of this study was to determine if the isolation frequencies of caries-associated micro-organisms (mutans streptococci, lactobacilli and yeasts) in 1 year old infants were associated with the isolation frequencies found in their mothers. Trained health visitors collected salivary samples (tongue loop) from 1051 infants and their mothers. Caries-associated micro-organisms were cultured and identified using standard laboratory methods. The lowest detection level for each of the taxa was 10^3 CFU/ml of saliva. Mutans streptococci (*Streptococcus mutans* and *Streptococcus sobrinus*) were recovered from 11% (when isolated: range 10^3- 10^6 CFU, median $5.00x10^3$) and 50%, lactobacilli from 5% and 40% and yeasts from 12% and 10% of infants and mothers respectively. If *S. mutans* were isolated from their mothers, there was an increase in isolation frequency in infants from 8% (when *S.mutans* was not isolated in their mothers) to 14% ($P<0.001$: chi squared test), for lactobacilli from 4% to 6% (not significant) and for yeasts from 11% to 21% ($P<0.001$). This observation would support the role of vertical transmission in the colonisation of specific bacterial taxa in infants mouths.

It is concluded that there were significantly higher isolation frequencies of *S.mutans* and yeasts (not lactobacilli) in infants if these bacterial taxa were isolated also in their mothers.

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Risk Markers for Future Caries in Infants

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The continued high prevalence of decay in Scottish 5-year-olds has been recognized as a priority area by the Scottish Office and targets were set for improvement. This project was part of a 4-year longitudinal study which aimed to identify risk markers for future caries in infants to allow targeted preventive care and promote a subsequent improvement in dental health. Of the 1974 infants born in Dundee between April 1, 1993, and March 31, 1994, 1,592 consented to participate in the study. The 57 Health Visitors in Dundee collected microbiological data (tongue-loop saliva sample) and social and medical data (parental and health visitor questionnaires) for the infants at 1, 2 and 3 years of age, respectively. Dental examination was carried out by the study dentist at the D_1 caries in enamel threshold. A risk model was derived from a χ^2 aided interaction detection analysis. 639 fully completed data sets from all sources for infants at 1 year were used to predict caries ($d_1mft>0$) at 3 years in these infants (incidence = 25%). Powerful predictors included housing type (owned/private/rented/social/other), food/drink at night (yes/no) and a health visitor assessment of caries risk (high/low). The model gave a sensitivity of 0.63 and a specificity of 0.53. These results indicate that certain markers at the age of 1 year can be used to predict future caries at the age of 3 years and a risk model derived from preliminary analysis may be used as a predictive tool for identification of high caries risk infants in a community setting.

181 Microbial markers for caries in infants. J.R. RADFORD*, Z. NUGENT, H.M. BALLANTYNE, C. LONGBOTTOM, N.B. PITTS, M. ROBERTSON AND D. BEIGHTON[1] (Univ. Dundee Dental School and [1]KCSMD, London, UK.)

The aim of the study was to determine if caries associated micro-organisms recovered from 1 year old infants (and their mothers) can predict caries development at 3 years of age. As part of a longitudinal study of caries markers, tongue loop saliva samples were taken from 1050 1 year old infants and their mothers (baseline). Mutans streptococci (*S.mutans*, *S.sobrinus*), lactobacilli and yeasts were recovered and characterised using standard microbiological techniques. Caries was diagnosed at the D1 (caries in enamel) level. Infants with caries at age 1 year (n=27) were excluded from subsequent analyses. Sensitivity and specificity (calculated at the D3 level) for the infants' bacteria were: total caries associated micro-organisms 0.30, 0.79, mutans streptococci 0.18, 0.91, *S.mutans* 0.18, 0.91, *S.sobrinus* 0.02, 0.99, lactobacilli 0.08, 0.96 and yeasts 0.10, 0.90. Sensitivity and specificity for the mothers' bacteria were: total caries associated micro-organisms 0.75, 0.34, mutans streptococci 0.56, 0.49, *S.mutans* 0.56, 0.50, *S.sobrinus* 0.08, 0.93, lactobacilli 0.54, 0.63 and yeasts 0.15, 0.90. In conclusion, oral microbiological markers in 1 year old infants, when used alone, are weak predictors for the presence of caries at 3 years of age. Other dental and social markers are needed to identify more reliably those infants at highest risk.

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Distribution of Caries monitored annually from ages 1 to 4 years in a Scottish Cohort

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The aim of this study was to monitor annually the distribution of caries within a cohort of Scottish children from ages 1 to 4 years. The sample consisted of 942 children participating in a longitudinal study to identify markers for caries in a cohort in Dundee, Scotland from 1994 to 1998 in a community setting. Caries was recorded at the d1 (enamel) and d3 (dentine) thresholds. The distribution of caries was calculated for: each of approximal and labial sites for incisors and canines (1- 4 years); at occlusal sites in 1st and 2nd molars (2 - 4 years); approximal sites in 1st and 2nd molars (3 & 4 years). Caries scores at ages 3 and 4 years were compared for those with and without labial caries at aged 2 years. The prevalence of caries for years 1 to 4 was : - d1mft>0: 2%; 12%; 28% and 49%; - d3mft>0: 0.3%; 4%; 12% and 33%. The prevalence of d1 labial caries in anterior teeth rose from 2% (yr.1) to 16% (yr.4). Approximal caries in incisors rose from 0.6% (yr.1) to 10% (yr.4). The prevalence of occlusal caries in the first molars rose from 1% (yr.1) to 29% (yr.4) and in second molars from 9% (yr.3) to 39% (yr.4). Approximal caries in molars rose from 3% (1st molars) and 0.4% (2nd molars) in yr.3 to 17% (1st) and 11% (2nd) in yr.4. For those children with labial caries in anterior teeth at aged 2 years the mean d1mft at 4 years was almost 4 times (6.6 vs 1.8) that of children without labial caries at 2 years and this difference was reflected in the level of caries in molar teeth.

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Caries-associated micro-organisms in infants from different socio-economic backgrounds in Scotland

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Abstract

Objectives: The aims of this study were: (1) to compare the frequency of isolation of mutans streptococci, (*Streptococcus mutans, Steptococcus sobrinus*), lactobacilli and yeasts (caries-associated micro-organisms) in the saliva of 1-year-old infants with and without dental caries; and (2) to determine if socio-economic background influenced the frequency isolation of bacteria and caries status.

Methods: 1393 1-year-old consented infants, who comprised 70.3% of children born in Dundee during a 1 year period, had saliva samples taken (tongue-loop method) for microbiological culture and were examined for dental caries (d_1-threshold: enamel and dentine diagnostic threshold). Thirty-nine infants were diagnosed with caries and the frequencies of isolation of caries-associated micro-organisms (and absolute microbial counts) were compared with infants who were caries-free. In addition, associations were sought between the infants' socio-economic background, the frequency of isolation of caries-associated micro-organisms and caries status.

Results: Streptococcus mutans, lactobacilli and yeasts were isolated more frequently from those infants with caries compared to those who were caries-free (*S. mutans*: 29.7 vs 9.8%, $P = 0.0008$; lactobacilli: 15.4 vs 4.3%, $P = 0.0073$; yeasts: 23.7 vs 10.4%, $P = 0.0016$—Fisher's exact test). There were no significant differences between the isolation frequencies of *S. sobrinus* (2.7 vs 1.3%, $P = 0.39$) from those with and without caries. Significantly, more infants living in areas of high deprivation had caries compared to those from more affluent areas (DEPCAT 6 and 7 vs 1–5: 3.6 vs 1.9%, $P = 0.049$), but, apart from yeasts, socio-economic background was not significantly associated with the isolation frequencies of any of the caries-associated micro-organisms.

Conclusions: In infants as young as 1 year of age, salivary *S. mutans*, lactobacilli and yeasts but not *S. sobrinus* were isolated significantly more frequently from those with caries compared to those who were caries-free. Apart from yeasts, socio-economic background did not influence the frequency of isolation of caries-associated micro-organisms. However, infants living in areas of highest deprivation had significantly higher frequencies of caries compared to those from more affluent areas.

Keywords: Mutans streptococci; Infants; Caries; Socio-economic

1. Introduction

There continues to be stark inequalities in the oral health of children with more than half the untreated tooth decay found in just 10% of 5 year-olds [1]. Identification of such children before caries has occurred would allow targeting of preventative care. A key putative marker is caries-associated micro-organisms and indeed many studies have reported correlations between mutans streptococci and caries status in adolescents (for key papers see [2–4]), but few [5–7] have looked for associations in infants, particularly as young as 1 year of age.

In addition, there is little information about relations between socio-economic status and caries experience in infants and, of those, no clear conclusion can be made [8–12]. There also appears no report in the literature examining the relationship between the frequency of isolation of mutans streptococci, lactobacilli and yeasts (caries-associated micro-organisms) and socio-economic status in 1-year-old infants.

As part of a 4 year longitudinal project with the aim of identifying markers for caries in pre-school infants, oral microbiological sampling and dental examinations were carried out on 1393, 1-year-old infants. Thirty-nine of

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these infants already had caries (when diagnosed at the d_1-threshold: enamel and dentine diagnostic threshold). The aims of this study therefore, were: (1) to compare the frequency isolation of caries-associated micro-organisms in infants with and without dental caries; and (2) to determine if socio-economic background was associated with microbial carriage and caries status.

2. Materials and methods

Health visitors carried out the oral microbiological sampling and a research dentist (HB) performed the caries examination. These data were collected in a 'field setting', viz. in the infants' homes, at health centres and at nurseries.

2.1. Subjects

The parents/guardians of all children (1981) born in Dundee, Scotland, during one calendar year (April 1993–March 1994) were approached for permission to allow their children to have microbiological samples taken, to be dentally examined and for other information to be collected including socio-demographic data. From a total of 1703 consented infants, salivary samples were taken from 1436, and 1419 were dentally examined. Salivary microbial and caries data were recorded for 1393 infants, who make up the group reported in this paper. Eighty-nine per cent of microbiological samples and dental examinations were carried out within a window of 3 months before, or 3 months after, the infant's first birthday.

The protocol for this non-interventionist designed caries-risk assessment study was approved by Tayside Committee on Medical Research Ethics.

2.2. Measure of relative deprivation or affluence

This is quantified in Scotland according to the method described by Carstairs and Morris [13]. In summary, scores (DEPCAT categories) are derived from 1991 Census data for populations in postcode sectors by combining the following variables: overcrowding, male unemployment, low social class and whether or not the household owns a car. The score is therefore a measure of a particular sector's socio-economic status relative to the average for Scotland, a score of DEPCAT 1 being the most affluent and 7 the most deprived. Such data can be obtained from the Public Health Research Unit, University of Glasgow, G12 8RZ (Carstairs Scores for Scottish Postcode Sectors from the 1991 Census, ed.: Philip McLoone).

In Dundee, only one child was born during the year in the DEPCAT 7 area and this single observation was included in DEPCAT 6.

2.3. Oral microbiological sampling and processing

The tongue-loop method [14] was used to collect the oral microbiological samples. Each sample was agitated into a vial containing 1 ml of LAB M Fastidious anaerobe broth (LAB M, Bury, England BL9 6AU), placed in a polystyrene block in a Combi Cold Carrier®, which was kept cool in a GIO'STYLE® cold picnic box (Jencons [Scientific] Ltd, Leighton Buzzard, England LU7 8UA) before transportation to a laboratory for microbiological processing.

Caries-associated micro-organisms were cultured and characterised as described by Beighton et al. [15]. In summary, samples were dispersed by vortexing for 10 s and mutans streptococci (*Streptococcus mutans and Streptococcus sobrinus*) were cultured on mitis salivarius agar plus 15% sucrose and 0.2 units per ml bacitracin, lactobacilli on Rogosa agar and yeasts on Sabourand Dextrose Agar (all media from Oxoid, Unipath Ltd, Basingstoke, UK). Plating-out was carried out within 4–8 h of collection. The mutans streptococci and the lactobacilli were incubated anaerobically and the yeasts were cultured aerobically for 3 days at 37°C.

After incubation, colonies with a characteristic morphology (*S. mutans*—raspberry-shaped and embedded into agar; *S. sobrinus*—again raspberry-shaped and embedded into agar but surrounded by a 'halo'; yeasts—white, flat and matt with a creamy consistency and a distinctive malodour; lactobacilli—the only colonies recovered on Rogosa agar) were counted, tested for catalase and Gram-stained. Confirmative identification of mutans streptococci was carried on a representative sample of presumptive isolates, based on a short set of biochemical and fermentation tests [16]. The lowest detection level for each of these taxa were 10^3 colony forming units per ml of saliva.

2.4. Caries diagnosis

A calibrated examiner (HB) carried out dental examinations on all the infants. Caries was diagnosed at the d_1 threshold according to the method described by Fyffe [17] based on a visual classification described by the World Health Organisation [18]. A pen torch (pen light) aided this 'field' examination.

2.5. Statistical analysis

Data were analysed using the SPSS® software package. The relationship of isolation frequencies of each of the bacterial taxa to caries status were examined using Fisher's exact test.

Microbiological data were also expressed as absolute counts of bacteria and Mann–Whitney U tests were used to look for differences between infants with caries and those who were caries-free. Receiver Operating Characteristic curve (ROC) analysis [19] was used to compare the relative efficacy of microbial counts as a diagnostic test for caries.

Associations between the degree of relative affluence or deprivation, as measured by DEPCAT, and caries and recovery of caries-associated micro-organisms were examined using the Mantel–Haenszel test for linear association. Because the majority of infants in Dundee live in areas of

Table 1
Caries-associated micro-organisms (% frequency isolation) in infants with and without caries

	Infants with caries	Caries-free	All infants	Fisher's exact test	Mann–Whitney[a] *U* test
Mutans streptococci	32.4	10.2	10.8	0.0003	0.0001
S. mutans	29.7	9.8	10.3	0.0008	< 0.0001
S. sobrinus	2.7	1.3	1.3	0.39	0.46
Lactobacilli	15.4	4.3	4.6	0.0073	0.001
Yeasts	23.7	10.4	10.6	0.0016	0.0066

[a] To look for differences in absolute microbial counts between those infants with caries and caries-free infants.

high deprivation, data from DEPCAT 1 and 2 were pooled for comparisons, as were the data from DEPCAT 6 and 7.

3. Results

Thirty-nine from a total of 1419 infants had caries. These 39 infants suffered from 116 decayed surfaces (94 enamel and 22 dentinal). Seventy-eight of the affected surfaces were on the upper incisors (only the upper and lower incisor teeth had erupted in the majority of children).

Isolation frequencies of caries-associated micro-organisms in infants with caries and those who were caries-free are shown in Table 1. Mutans streptococci were isolated significantly more frequently from those infants with caries compared to those who were caries-free. The mutans streptococci were comprised almost entirely of *S. mutans*. Similarly, lactobacilli were isolated significantly more frequently from those infants with caries compared to those with no caries, as were yeasts. This was in contrast to *S. sobrinus* where there were no significant differences between the groups. Regardless of caries status, mutans streptococci, *S. mutans*, *S. sobrinus*, lactobacilli and yeasts were isolated from 10.8, 10.3, 1.3, 4.6 and 10.6%, respectively.

When absolute microbial counts were analysed, similar differences were found between those infants with caries and those who were caries-free (Table 1). Receiver Operator Characteristic curves (Fig. 1) showed that absolute microbial counts were not a good surrogate diagnostic test for caries.

The degree of relative affluence or deprivation did not significantly influence the proportion of infants who harboured mutans streptococci and lactobacilli except for yeasts, which were associated with greater deprivation (Table 2) This association of yeasts with deprivation was maintained when infants with $d_1t > 0$ are excluded from the analysis.

Those infants living in areas of greatest deprivation had significantly higher mean caries (DEPCAT 6 and 7 vs 1–5: 3.6 vs 1.9%, $P = 0.049$).

4. Discussion

This study has demonstrated that in Dundee, Scotland, 1-year-old infants with caries have higher isolation frequencies and higher counts of *S. mutans*, lactobacilli and yeasts but not *S. sobrinus* compared to those who were clinically caries-free. Similar findings have been published by Grindefjord et al. [20] who reported that mutans streptococci and lactobacilli were significantly associated with caries in 2.5-year-old children (over half were categorised as 'children with immigrant background') living in Stockholm. In addition, numbers of mutans streptococci have been shown to be significantly correlated with caries prevalence in 365 1- and 2-year-old Japanese infants [7]. These results would appear to contrast with those reported by Matee et al. [21] who found there were no differences in the isolation frequencies

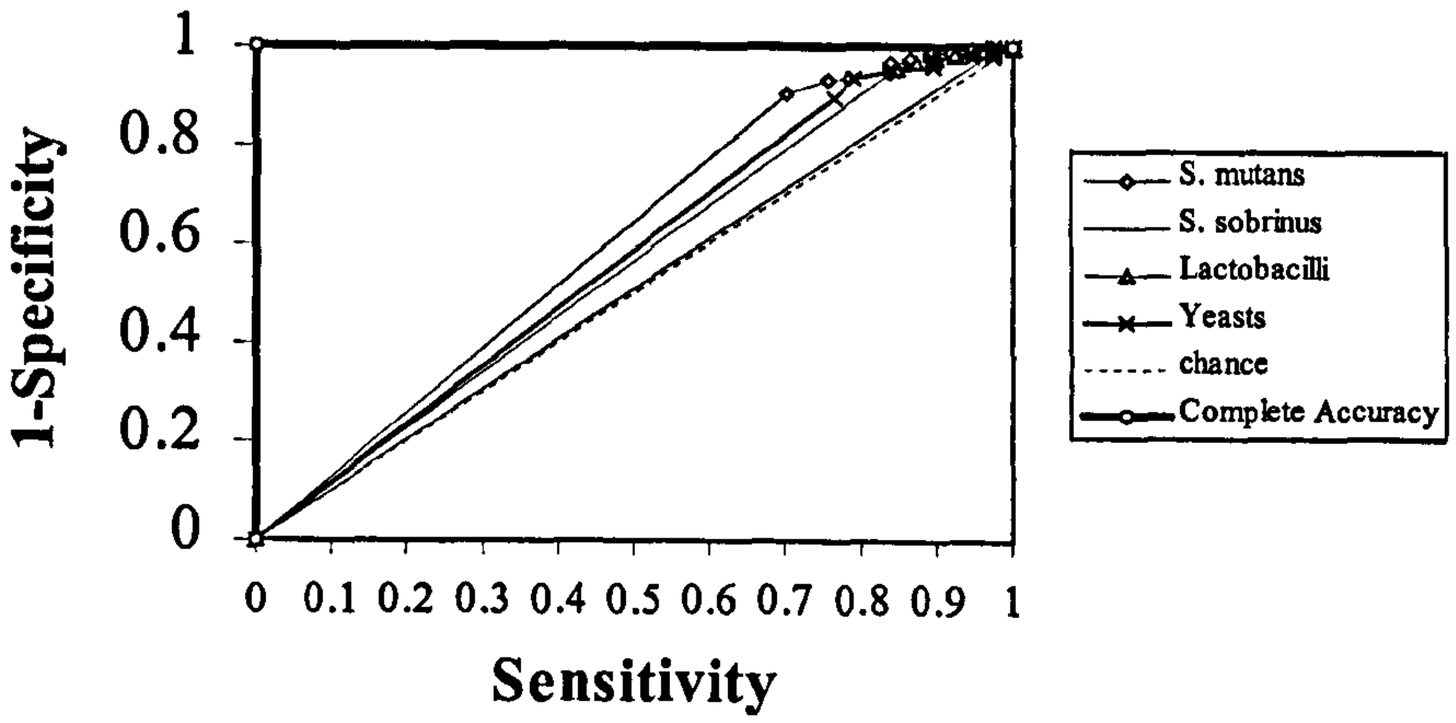

Fig. 1. Receiver–Operator Characteristic (ROC) curves for microbial counts.

Table 2
Associations between (1) caries and (2) the recovery of caries-associated micro-organisms and DEPCAT

	DEPCAT					P (all infants[a])	P ($d_1t > 0$ excluded[b])
	1 and 2	3	4	5	6 and 7		
No. of infants	318	99	188	66	722		
% dmft > 0	0.6	1.9	2.6	6.2	3.6	0.005	–
% Mutans streptococci	8.6	8.7	13.6	12.1	11.1	0.26	0.43
% *S. mutans*	8.0	7.8	12.6	12.1	10.9	0.14	0.23
% *S. sobrinus*	1.2	1.0	1.6	0	1.4	0.87	0.95
% Lactobacilli	2.7	6.8	3.6	4.4	5.1	0.15	0.10
% Yeasts	7.8	10.8	9.9	9.2	12.3	0.039	0.041

[a] Mantel–Haenszel test probabilities for all infants.
[b] Mantel–Haenszel test probabilities if infants with $d_1t > 0$ are excluded.

of *S. mutans* (plaque samples) between caries-active and caries-free 1–3.5-year-old infants from regions of Tanzania with high and low prevalences of caries. It would appear, however, that although there is a correlation between caries and mutans streptococci in industrialised countries, this may not be generalisable for young children in non-industrialised countries such as those on the African continent [22].

Isolation rates for mutans streptococci in infants vary dramatically between studies. In this study, 10.8% of infants harboured mutans streptococci which is comparable to that reported by Grindefjord [23] who recovered mutans streptoccoci from 6% of a group of 1095 1-year-olds. These results are in contrast to other studies, in similar age groups, which have reported isolation frequencies varying from 43% [24] to circa 90% in Canadian children [25]. A possible explanation for such variation could be methodological differences arising from sampling and culture. However, it has been reported [22] that the viable counts of mutans streptococci obtained using the spatula method, the tongue-loop method, commercial dip-slide methods and others are all significantly correlated with the counts obtained using conventional paraffin-stimulated saliva cultured on selective media. It would therefore appear that methodological differences alone would not account for such variation. Another possible explanation is the difference in sugar consumption between the various demographic groups of children, although the relationship between *S. mutans* and diet in the population is far from clear [4,26,27], as many studies have been carried out under abnormal or extreme conditions [28].

Mutans streptococci were recovered from only one-third of infants with caries. It maybe that seeding of bacteria from d_1 lesions into saliva is not very effective or alternatively the numbers of mutans streptococci were less than the lowest detection level (10^3 colony forming units per ml of saliva).

Few studies have reported the frequency of lactobacilli in pre-school children. In our study, lactobacilli was recovered from 4.6% of infants which is less than but comparable to that reported by Roeters [29] (11.5%) who sampled 252, 2-year-olds at baseline in a 3-year cohort study and 13.1% in 36–47-month-old Caucasian children in a study [30] which compared caries prevalence and caries-associated micro-organisms in Caucasian and Afro-Caribbean children. This is in contrast to the results reported by Kohler et al. [31] who found that approximately 40% of 3-year-old children carried lactobacilli in their saliva.

Streptococcus sobrinus was isolated from only 1.3% of infants. This was not unexpected as this species was only recovered from 7% of 5–8-year-old Hampshire schoolchildren implying a low prevalence in the UK [32].

In contrast to the smaller sample sizes employed in many studies, in the present study 1400 children were examined thereby minimising the effect of individual variation. In addition, microbiological sampling and dental examinations were carried out within 3 months of the infant's first birthday, reducing the effect of age on the increased acquisition of oral bacteria [33] and caries increments. Few studies have reported the caries prevalence in 1-year-old children although recently two major studies [11,12] have been published. Four per cent of children from the UK aged 1.5–2.5 and 6.4% of 1-year-old children from the US had caries experience at the d_3 (caries into dentine) level. The present study found that 2.7% of infants were suffering from dental caries, which is within the same range as that reported by Wendt et al. [34] who studied 632, 1-year-old Swedish children (again using the d_3 diagnostic threshold).

In the present study, caries was diagnosed at the d_1 level. Ideally, in order to examine caries at this threshold, teeth should be dried before examination. It was decided to carry out the caries examinations without an airline to dry the teeth as it was anticipated that this could lead to infant distress, reducing the acceptability of the examination. Performing the dental examination in a 'field setting' allowed us to access more children.

Seventy eight per cent of carious surfaces diagnosed in this study affected the upper incisor teeth which was not surprising as, at 1 year of age, generally only the upper and lower incisor teeth are fully erupted with the first lower deciduous molars partially erupted. Grindefjord et al. [20] reported that 72% of carious lesions in their study were localised to the maxillary incisors of 2.5-year-old children. They suggested that this might partly be explained by

a higher intake of sugar-containing beverages at night. This poses the vexing question as to whether or not a significant proportion of caries observed in under 2-year-olds is in fact nursing caries (early childhood caries—ECC [35]). It should be pointed out, however, there is much confusion surrounding the prevalence and epidemiology of ECC, some questioning as to whether indeed this is a distinct entity only distinguished by the generalised distribution of the lesions.

Krasse [36] paradoxically referred to those children with more than 10^6 *S. mutans*/ml of saliva and at particular risk from dental caries, as 'millionaires'. This was based on a study of a large group of 645, 9–12-year-old children who showed that those with these high numbers of *S. mutans* and lactobacilli developed significantly more carious lesions after 1 and 2 years compared to children with low counts. We used ROC analysis [19], to determine if such a relationship could be found in 1-year-olds. Although microbial levels are related to caries status, they did not constitute a good screening test and this was not unexpected as a stark microbial threshold is probably a gross simplification of the carious process [37].

There do not appear to be any papers in the literature that have reported associations between socio-economic status and the recovery of caries-associated micro-organisms in 1-year-old infants. Apart from yeasts, this study was not able to demonstrate any such relationship. This was not unexpected as few teeth had erupted in 1-year-olds and, of those present, there had been insufficient time for the establishment of an amphibiont microflora.

However, it was found that those infants, even as young as 1 year of age, living in areas of high deprivation had a greater experience of caries compared to those from more affluent backgrounds. This supports the findings of a multistage random probability design study [11] analysing data derived from The National Diet and Nutrition Survey of UK children aged 1.5–4.5 years, which reported that caries was found to be most strongly related to receipt of income benefit, educational status of the mother and social class of the head of the household. Interestingly, this simple relationship was not found in 2-year-old children from the Riyadh region of Saudi Arabia [8], 1- and 2-year-olds from Goiânia-GO, Brazil [9] and school children living in the inner city are of Camden, London, UK [10] (in contrast to ethnicity in this latter study). Roeters [38] reported that the correlation between socio-economic background and caries becomes stronger with increasing age (as also reported in groups of older children in Riyadh and Goiânia). Despite a high proportion of our sample lived in areas of high deprivation (the Arizona preschool study [12] purposely oversampled children from low-income backgrounds and therefore were not able to look for such an association), the size of our group enabled us to demonstrate a relationship between socio-economic background and caries that could not have been shown in a smaller experimental group.

One of the key questions we aim to answer is whether those pre-school infants who harbour caries-associated micro-organisms but who do not have caries at 1 year of age are at increased risk from future caries compared to those who do not carry these microbial taxa. If such a relationship could be demonstrated, microbiological sampling of saliva to identify pre-school infants at risk of future caries offers a strategy for targeting preventive care at these infants. Analysis of longitudinal data will seek to answer this question.

In conclusion therefore, this study has shown that *S. mutans*, lactobacilli and yeasts were isolated significantly more frequently from the saliva of 1-year-old infants with caries compared to those who were caries-free. Socio-economic background did not influence the frequency of isolation of caries-associated micro-organisms apart from yeasts, although those infants living in areas of high deprivation had significantly more caries than those from more affluent areas.

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Anecdotes

The author suggests this thesis would be incomplete on omission of the following anecdotes. Collected during the four-year study duration, they should serve as a reminder to those entering the field of epidemiological research, especially home visiting.

'Always expect the unexpected'

A – 1 The 'carpet-eating' child

A – 2 The 'poacher' father

A – 3 The 'horse' mother

A – 4 The 'mutant' study

A – 5 Photographs of a typical home visit – no one home!

A – 6 List of experiences of home visiting

lina Din
Wedderburn Street
ndee

11930227

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Study Number

HEALTH VISITOR/MUTANS STUDY

AL / MEDICAL / ORAL INFORMATION SHEET
(2 YEARS)

Date: 27/10/95.

e Marie Buchan

inic ☐ Other ☐

? 2.10.

the child at high risk of developing dental decay? ~~yes~~ / no

		Centile		Centile		Centile
Weight at 2 years	? 9 kgs	33rd	Height at 2 years	81 cms	10th	O.F.C. at 2 years	47 cms	46th

Immunisation status ..... Up to date .....

Does child suffer from any illness(es) requiring long term medication? ~~yes~~ / no

If yes, nature of illness and medication .....

.....

Use of dummy/comforter? ~~yes~~ / no. If yes, still being used? ~~yes~~ / no

Are vitamin supplements given? yes / no

Feeding problems of significance? ..... Poor eater – Eats rubber teething aff carpet

Number of siblings? 5.

Mother's employment part-time / full-time / none Nature of employment .....

Married / Single / Cohabiting / Living with parents?

Father's employment / past employment none .....

Parents Health - any significant data?

Mother ..... ~~Smoker~~ / Non smoker?

Father ..... ~~Smoker~~ / Non smoker?

Housing: Owner / Occupier ☐ Private Rent ☐ Council / SSHA Rent Council

Updated address

If moved in last 12 months :

If you have any relevant additional information please add this overleaf.

ns/general/qulvyr2

1405-1301-F1

Study Number

HEALTH VISITOR/MUTANS STUDY

CONFIDENTIAL

SOCIAL / MEDICAL / ORAL INFORMATION SHEET
(2 YEARS)

To be completed by Health Visitor

Date: 5 / 6 / 95

Health Visitor's name: E. TILBURY

Number of teeth present at 2 years? 20

Health Visitor's Assessment : Is the child at high risk of developing dental decay? yes / ~~no~~

		Centile
Weight at 2 years	13	50th
Height at 2 years	90.25	90
O.F.C. at 2 years	50	9

Immunisation status ..... Complete .....

Does child suffer from any illness(es) requiring long term medication? ~~yes~~ / no

If yes, nature of illness and medication .....

.....

Use of dummy/comforter? yes / ~~no~~ If yes, still being used? yes / ~~no~~

Are vitamin supplements given? ~~yes~~ / no

Feeding problems of significance? ..... No .....

Number of siblings? 1

Mother's employment part-time / ~~full-time~~ / none Nature of employment .....

~~Married~~ / Single / ~~Cohabiting~~ / ~~Living with parents~~?

Father's employment / past employment /none ..... Poacher .....

Parents Health - any significant data?

Mother ..... Back ..... ~~Smoker~~ / Non smoker?

Father ..... ~~Smoker~~ / Non smoker

Housing: Owner / Occupier ☐ Private Rent ☐ Council / SSHA Rent ☑

Updated address

If moved in last 12 months :

32 CRAIGMORE STREET.

If you have any relevant additional information please add this overleaf.

0109930185

Study Number

HEALTH VISITOR/MUTANS STUDY

CONFIDENTIAL

SOCIAL / MEDICAL / ORAL INFORMATION SHEET
(2 YEARS)

To be completed by Health Visitor

Date: 17/05/95

Health Visitor's name: ELEANOR FRASER

Number of teeth present at 2 years? 20

Health Visitor's Assessment : Is the child at high risk of developing dental decay? yes / no

		Centile		Centile		Centile
Weight at 2 years	12kg		Height at 2 years	87.5	O.F.C. at 2 years	47.5	

Immunisation status ..... FULLY IMMUNISED .....

Does child suffer from any illness(es) requiring long term medication? yes / no

If yes, nature of illness and medication ..... NIL .....

.....

Use of dummy/comforter? yes / no. If yes, still being used? yes / no

Are vitamin supplements given? yes / no

Feeding problems of significance? ..... NIL .....

Number of siblings? NIL

Mother's employment part-time / full-time / none Nature of employment ..... HORSE .....

Married / Single / Cohabiting / Living with parents?

Father's employment / past employment /none ..POST GRAD RESEARCH ASSISTANT .....

Parents Health - any significant data?

Mother ..EAR + THROAT INFECTIONS (FREQUENTLY) Smoker / Non smoker?

Father ..... Smoker / Non smoker?

Housing: Owner / Occupier ✓ Private Rent Council / SSHA Rent

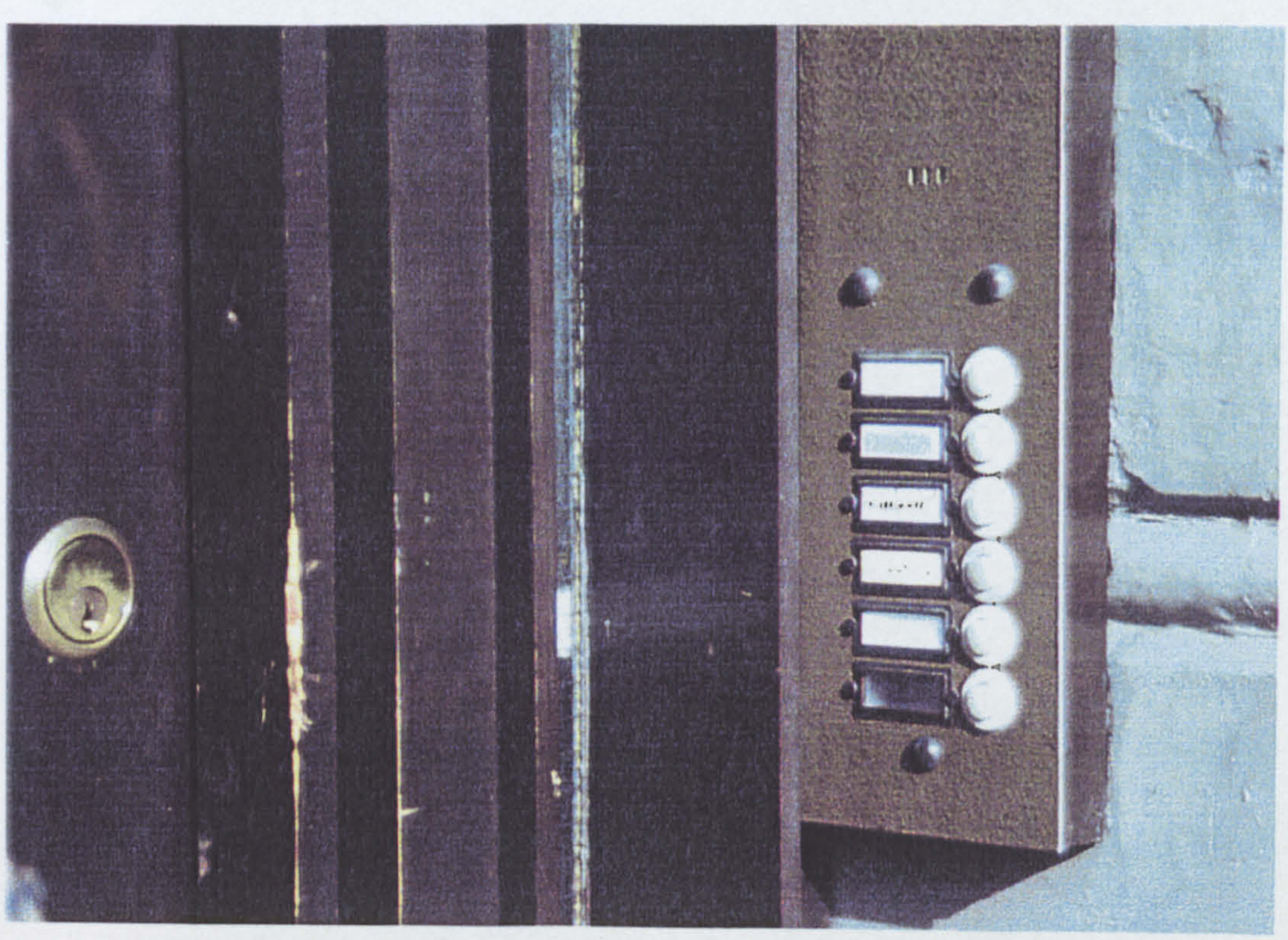
Updated address

If moved in last 12 months :

If you have any relevant additional information please add this overleaf.

Mutans/general/quhvyr2

Dental mutant Study



Anecdotes: Photographs of a home visit by the study dentist.

A – 6 List of experiences of home visiting

1. Examination of one child under the persistent glare of a very protective doberman named 'Satan' sitting close by on the sofa

2. Asked by one mother, "so, do you *know* anything about teeth then?"

3. Requested by a child, "you know you can't go without a kiss"

4. Abandoned suddenly by a mother who maintained that her son had to have sweets from the ice-cream van before I could look at his teeth.

5. Being handed £10-00 from a father as he opened the door. Seeing my quizzical look he said, "you are the milkman aren't you?"

6. Response from a mother after answering door, "ye dinnae look like a dentist tae me". She then went on to clarify, "yer no a' auld an wrinkly like, ken"

7. Request from a mother, "you don't mind seeing him in the bath do you? You don't have to look at his Dad"

8. Informed by a mother, "he's got a mouthful of fish - actually he's had it for an hour but you can give it a go if you like"

9. Reply from a 4-year old, "I'm glad I've got holes in my front teeth because it means I can whistle"

10. Whilst walking through a nursery school a wee boy exclaimed loudly, "hey missus, you're huge!"

11. On examining a 4-year old in one particular nursery, one wee lad said to another, "'*teethies*', how old does she think we are?"

12. Request by a father, "do you think you could have a look at the spots on his head while you're here?"

13. On visiting a large house occupied by a number of families, a woman asked which child I was there to see. On my reply she systematically picked a number of children out of a room one by one until she had located the correct one.

14. Request from a father, “can you have a look at me as well, I’ve been up all night in agony”

15. One little girl said to her mother, “mummy, mummy, why has the dentist got clothes on?”

16. After an interesting conversation with a mother who asked me for my opinion on fluoride, it emerged she was a local councillor – *against* water fluoridation.

17. Request from a mother, “could you have a look at my puppy while you’re here and see if his second teeth are coming through okay”

18. One mother said, “could you just wait a minute” and left her daughter and Barney, the bearded collie, in my care while she nipped out to have a word with the man fixing the neighbours bathroom window – for 15 minutes!

19. To the question, “are you cohabiting?” asked by the health visitor, one mother replied, “aye, ah get it twice on a Tuesday noo”

20. Questions and advice from mothers of children with decay:

- Do you think it's something in the water?
- She never gets sweets (even though child is eating one)
- They grew in like that – just like her father's
- It was after he had the cold – they just all turned black
- It was that Farley's no added sugar that caused the rot (as her 2-year old ate a bowl of Dolly Mixture)

21. General advice given to me by several health visitors:

- Watch out for condoms on your exhaust pipe
- they can blow up to quite a size before bursting.
- Never use your finger to press the button in lifts – and don't lean on the walls either.
- Watch out if you're kneeling on floors (a change of trousers is a handy thing in the car).

- Buzzer systems are designed to keep you out and to allow the response, "but you never called"
- You always know the house you've to visit - it's the one with all the dandelions in a row of nice gardens and always the last flat to be occupied in a condemned block.
- Don't take you shoes off unless you're asked.
- Have a wee look through the letterbox to check the house is actually occupied but make sure you understand about letterboxes. You see there's ones with dogs behind, ones with brushes behind designed for skinning knuckles and there's always ones covered with sellotape to keep the draught out (and your calling slip).

And beware when taking a wee peep through - a metal-backed letterbox is guaranteed to fool you into thinking someone is staring right back at you!